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GUEST EDITORIAL

Financing Medical Education

THE financial status of our medical schools is of major concern to educators and the medical profession and should be of concern to the general public. Rising costs, which have not been balanced by proportional increases in endowment or appropriations, have forced curtailment of essential activities. It is becoming difficult to attract and hold salaried faculty members. Demands for expansion of enrollment are unrealistic unless accompanied by relative increases in staffs and facilities.

The solution to these problems is not to be found in further increases in tuition fees, which are already so high that there is danger of excluding from the medical profession talented young men and women whose financial resources are limited. It has even been proposed that the medical student bear the entire cost of his education, either by cash payment or notes payable after he enters practice. Why the physician, and not the banker, lawyer or engineer, should be expected to pay full costs is difficult to understand.

During the past few years, support for medical research has improved as a result of increased donations to national health organizations, federal appropriations and income from endowments of foundations. The availability of these funds, however, has not made the financial problem of the medical schools more simple because they have contributed to, rather than diminished, the basic costs of operation. The need is for "hard money" which can be depended upon for faculty salaries and basic operating expenses.

One of the most encouraging prospects is the almost simultaneous development of two organizations, the American Medical Education Foundation and the National Fund for Medical Education. The former, sponsored by the American Medical Association, and the latter, sponsored by prominent business men and educators, are combining their efforts to obtain assistance on a voluntary basis. During the first year of operation, $1,594,373 was distributed among the 79 schools. Such funds can be used without restriction to meet immediate requirements. They offer an opportunity to physicians to repay the investment of endowed or public funds made in their education without compulsion and in proportion to their means.

Contributions from physicians are made directly to the American Medical Education Foundation and may be designated for distribution to a specific school. Undesignated funds are allocated on an equitable basis and earmarked contributions are added to the basic grants. Thus physicians may receive credit from the schools of their choice for gifts to the Foundation. The administration of the Foundation is underwritten so that every cent contributed goes to a medical school.

Contributions from industrial corporations are routed through the National Fund

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for Medical Education. Quoting its by-laws, "The purpose of the Fund is to raise, dispense and administer funds, and to take other appropriate action, to promote and foster the following objectives:

(1) The interpretation of the needs of medical education to the American public.

(2) The encouragement of the growth, development and advancement of constantly improving standards and methods in the education and training of all medical manpower in the nation.

(3) The preservation of academic freedom in the institutions of medical education and the aiding of these institutions in offering equality of educational opportunity to all those who are qualified to seek to enter the medical profession.

(4) The strengthening of the nation's ability to survive by training and educating a sufficient corps of well-equipped men and women to care adequately for the people's health and medical needs."

When a group outside of medicine sets out to achieve such objectives, it deserves the support and encouragement of those who are more intimately acquainted with health problems. The practicing physician, in his daily contacts, can be of tremendous assistance in advancing the objectives of the National Fund.

As the prospect of solving the problem entirely by voluntary contributions seemed remote, the medical schools, through the Association of American Medical Colleges, went on record in support of federal aid to medical education. In 1951, 68 of the 79 schools voted in favor of legislation which would provide subsidies for operating expenses in proportion to enrollment, with no strings attached. They voiced serious opposition to any form of federal subsidization which would entail governmental interference in selection of students, size of enrolment, appointment of faculty, arrangement of curriculum or other internal policies which must remain under jurisdiction of their local governing bodies.

The American Medical Association has gone on record in opposition to federal aid for instructional expenses, in spite of generally favorable experience with federally administered research grants. It is their contention that federal subsidies should be discouraged until all other sources have been exhausted and until it can be demonstrated that federal aid can be provided without federal control.

The ultimate solution is not yet in sight. Although financing by endowment, voluntary contributions and taxation at the state or municipal rather than the federal level would probably be favored by the majority of the medical schools, they are reluctant to sacrifice the high standards of medical education while awaiting the day when their needs may be recognized locally. Whether or not federal aid is made available in an acceptable form, it is certain that a hard core of funds from other sources is essential.

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REPORT OF THE 1951 RABIES OUTBREAK RICHMOND, VIRGINIA

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Rabies in dogs has occurred in varying areas in Virginia since Colonial times. Until 1951 Richmond fortunately had relatively few cases of rabies, in spite of its large stray dog population. Even when an outbreak occurred in an adjacent county in 1950, no cases were known to have developed in the City.

This freedom from rabies came to an end with an occurrence of a rabies epizootic in the City which began on July 13, 1951, and lasted until September 9 of the same year. During this time, 29 animals (19 dogs and 10 cats) were shown by laboratory examination to have rabies; 5 others were designated as suspicious. The outbreak started on the far east side of town, which was immediately placed under quarantine. It spread by short jumps and intervals north and west until the entire portion of the City north of the James River had to be quarantined. When no cases appeared during the three weeks after September 9, the quarantine was raised. In the ensuing six months, no further cases of rabies have occurred.

TABLE I

CASES OF RABIES IN ANIMALS, STATE OF VIRGINIA, CITY OF RICHMOND, and ADJACENT COUNTIES DURING THE TEN-YEAR PERIOD 1942-1951.

	Virginia	*Richmond*	*Chesterfield County*	*Henrico County*
1942	89	0	0	0
1943	277	0	0	0
1944	334	0	*	*
1945	116	0	0	0
1946	108	0	2	0
1947	162	2	0	1
1948	149	0	0	0
1949	82	0	0	1
1950	94	0	11	2
1951	223	29	0	0
TOTALS	1,634	31	13	4

*—Not available

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HISTORY

Rabies has been known since ancient times and, in the main, has been associated with the genus canis. Rabies in dogs and domestic animals was described in 500 B. C. by Democritus and by Aristotle in 322 B. C. The relationship of hydrophobia in man to rabies in animals was recognized by Celus in 100 A. D., who advocated cauterization of wounds produced by rabid dogs.

Rabies in Western Europe antidates 1271, being prevalent among wolves in France at that time. The first recorded epizootic of this disease among urban dogs occurred in Italy in 1708 and later was reported in England in 1734. There is no evidence to indicate the existence of rabies in the New World prior to colonization. The first record is from Virginia referring to the disease in 1753.

The modern concept of rabies was originated by Pasteur and his associates, who theorized that the infective agent was sub-microscopic and called it a virus. This fact was established in 1903 by Rembinger. Pasteur developed a means of modifying the pathogenicity of the rabies virus and from this work developed a method of vaccination against the disease. On July 6, 1885, a boy, Joseph Meister of Alsace, became the first human treated by vaccination.

CONTROL OF OUTBREAK

The control of the outbreak of rabies in Richmond in 60 days may have been, in part, fortuitous; however, the prompt and thorough co-operation of the public in the involved area and the untiring efforts by the personnel of the City Dog Pound, the Game Warden, the Police Department, and the Society for the Prevention of Cruelty to Animals undoubtedly were the major factors in the effectiveness of the control measures. During the quarantine period, 622 dogs and 281 cats were destroyed as strays.

OBSERVATION OF ANIMALS

All animals suspected of having rabies, or impounded because of biting humans, were kept under quarantine for 14 days and regularly observed by the City Veterinarian for symptoms of the disease.

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Any dog dying with symptoms suggestive of rabies, for no apparent reason, or in less than 10 days after having bitten a human, was further examined for laboratory evidence of rabies by microscopic examination of its brain tissue and by mouse inoculation tests.

Many of the dogs developed clinical symptoms which could be classified as either typical furious rabies or dumb rabies. The dog with furious rabies, though previously of a quiet disposition, develops a tendency for wildness, leaving home, and running at large. In dumb rabies the animal, usually active and friendly, develops lassitude, tends to hide, refuses food, and his normal friendliness is replaced by a tendency to snap at people. Both conditions in the late stages produce paralysis of the hindquarters and some partial paralysis of the jaw muscle, frequently resulting in slavering. Progressive generalized paralysis and coma usually precede the animal's death.

In addition to the two types of rabies described above, during the outbreak there were some animals which died suddenly with no symptoms noted. This occurrence has been described previously by Rivers.[1]

LABORATORY TECHNIQUE

The spread-smear technique was used for making the preparation for microscopic examination in the Richmond Health Department Laboratory, and Seller's Stain was the stain of choice. This spread-smear technique is that described in Communicable Disease Center Bulletin,[2] and in the "Manual of Routine Diagnostic Methods of the Bureau of Laboratories of the Alabama State Department of Health".[3] The advantage of this method is that larger amounts of material may be examined per slide than with the other popular method known as the impression technique. Also, much more time per slide can be spent in the close scrutiny of each preparation. The disadvantage is that there is often damage to the neurones and glial structures and extracellular inclusion bodies are more common.

However since 1903, when Negri originally described the typical inclusion bodies of rabies that bear his name, there has been a trend to report as positive any slide with typical inclusion bodies showing dark inner granules. Gradwohl states: "In most preparations, many of the Negri bodies are not in the cytoplasm."[4] In the Manual of the Alabama State Laboratories mentioned above is found the following statement: "A positive report may be made on characteristic Negri bodies outside the cells, but search should be made for intracellular bodies."[3]

Other authorities make similar statements. Topley and Wilson say: "Negri bodies are found most frequently in the cytoplasm and dendritic prolongations of the nerve cells, particularly in those of Ammon's Horn."[5] Cameron goes further to state: "The finding of Negri bodies constitutes a positive diagnosis; however, suspicious bodies found in fresh brains should be considered as probable evidence of rabies."[6]

The Negri bodies observed by the method used were small, round or oval bodies, varying in size from 1.0 to 30.0 microns, and were of a homogenous magenta color with dark blue to black basophilic inner granules. While they were seen most frequently as extracellular, due to the method of preparation of the specimens, it was possible to find intracellular bodies if the slides were exhaustively searched. In at least 50 per cent of the slides, Negri bodies were found intracellular as well as extracellular. In all but 5 animals reported as positive for rabies, intracellular or extracellular Negri bodies were found. The 5 reported as suspicious were from animals in which atypical inclusion bodies were noted on examination.

There occurred during this rabies outbreak some cases of distemper. While many of these were confusing as to diagnosis from the clinical standpoint, the laboratory findings were clear cut. Examination of these animal heads revealed typical distemper inclusion bodies which, although somewhat resembling the Negri bodies of rabies, were easily differentiated. If present, they were invariably more numerous and never presented any suggestion of dark-colored granules. In no slides examined during this period did there occur both types of inclusion bodies.

All slides designated as positive were checked by at least two other individuals one of whom had to be the laboratory director or his assistant. Both of these individuals have had considerable training in the interpretation of such slides in other laboratories, one in the Alabama State Department of Health, and the other in the Virginia State Department of Health Laboratory.

In addition to the microscopic examination of the

brain tissues of infected or suspected animals, a further diagnostic procedure is available. This is the mouse inoculation test. Material from the brain of the suspected animal is inoculated intracerebral into 10 white mice. If rabies existed in the animal under suspicion, the mice generally succumb to the disease, and Negri bodies may be found in their brain material.

MOUSE INOCULATION

Ordinarily when the microscopic examination reveals Negri bodies, no further studies are done on the animal, as this is considered sufficient evidence of rabies. However, due to interest in evaluating the findings by mouse inoculation of material from all suspected animals, portions of all negative brains, and 22 of those found to be positive, were sent to the Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C. Insofar as rabies was concerned, all of these specimens gave negative results by mouse inoculation.

Feeling that this was due in part to the delay encountered in transportation and shipping, the Richmond Health Department laboratory began to do its own mouse inoculations. This began, however, towards the end of the outbreak, with only two positive animals included in this group. None of the inoculated mice developed signs of rabies.

It is difficult to explain why the mice inoculated with material from the two animals which were observed positive by microscopic examination did not acquire the disease. In spite of the negative results on mouse inoculations, microscopic examination alone would be acceptable proof of the existence of rabies. Nevertheless, this does show the need for closer cooperation between the field and the laboratory and the use of some type of refrigeration, even when no shipment problem is involved.

A SECOND ENTITY

There is a further possible complicating factor which, at this time, is in the investigation stage. During an outbreak of rabies in St. Louis, where they have a complete virus laboratory at their disposal, it was discovered that concurrent with the typical rabies another entity apparently was occurring. This second entity caused development of Negri bodies which were observed by microscopic examination of the dog brain, but it could not be demonstrated to be pathogenic for mice.

Mice inoculated with such material, instead of dying of typical rabies on the tenth day after inoculation, either survived with no abnormal findings being noted or developed an encephalitis-like condition on or after the 21st day. This entity, except for showing no cross immunity with rabies, could not be identified. While other laboratories have noted such an occurrence, there is only one brief and indirect reference to such an occurrence in the literature, and this is in a transcript of a clinical pathologic review held at the Washington Medical School in St. Louis.

In spite of this theoretical entity, nearly all Public Health laboratory workers hold that it would be a dangerous practice to fail to report as positive for rabies any microscopic examination which showed typical Negri bodies. When reporting test results in a disease wherein the mortality rate is known to be 100 per cent, one cannot afford to take chances or even to delay treatment in the face of such findings by waiting for further investigation by animal inoculation which may or may not be conclusive. Therefore, it was not deemed advisable in this outbreak to disregard direct microscopic findings regardless of results of animal inoculations.

CONTROL PROCEDURES

"The Control of Communicable Diseases in Man", the report of the Committee on Communicable Disease Control of the American Public Health Association which report was officially approved by the United States Public Health Service, and was later, 1949, formally adopted by the Board of Health of the City of Richmond, and by the Virginia State Board of Health in 1950, discusses the disease entity rabies in detail. The report contains the following explicit statements and facts concerning rabies control procedures:

"B. The infected individual, contacts and environment:

1. Recognition of the disease and reporting: Clinical symptoms, supported by the presence of Negri bodies in the brain of the animal which has caused the injury, and by animal inoculations with material from brain of such animal.
2. Isolation: None if the patient is under adequate medical supervision, and immediate attendants are warned of possibility of inoculation by human virus through contamination with the patient's saliva.
3. Concurrent disinfection: Of saliva of patient and articles soiled therewith.
4. Terminal disinfection: None.

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5. Quarantine: None.
6. Immunization: Prompt institution of prophylactic antirabic vaccination of persons bitten by or intimately exposed to the saliva of a rabid animal, or any animal suspected of being rabid, especially a dog. Treatment, if not completed, may be discontinued if the dog is well at the end of the period of observation, or rabies is excluded by suitable laboratory examination. Chance of infection is to be weighed against the very small chance of developing paralysis due to the treatment.
7. Investigation of source of infection: Search for the rabid animal and for all persons and other animals bitten by it.

C. Epidemic measures:

1. Establishment of area control under the authority of state laws and regulations enforced by state and local health authority, or state department of agriculture, or live stock sanitary board.
2. Within defined area of epidemic prevalence of rabies in wild or domestic animals or both, vigorous and sustained efforts should be made to collect and destroy all stray, ownerless, and un-vaccinated dogs found off the owners' premises.
3. Use of appropriate means of educational publicity to advise the public of the local rabies situation, to inform them of the nature of the disease, its severity, the means of transmission, the precautions they should take regarding their own dogs and those of others with which they may come in contact, the necessity of reporting to a physician for treatment any person bitten by a dog. The presence of a dog showing unusual, strange, or threatening behavior should be reported to the police.
4. Special encouragement of and facilities for the protection of owned dogs by vaccination.
5. All cases of rabies in man or animals and all dog bites of persons should be reported promptly to the local health authority."[7]

STATE RECOMMENDS THREE-POINT PROGRAM

The Virginia State Health Commissioner has pointed out that, at present, control of rabies is chiefly a matter of local legislation, planned and enforced by local officials. He recommends the following three-point control program:

1. Impounding and destruction of all stray and ownerless dogs.
2. Vaccination of all dogs.
3. Licensing of all dogs.

TREATMENT OF PERSONS EXPOSED TO RABIES

As a result of the Richmond outbreak, there were 88 persons who were given anti-rabies treatment. Among this number were some individuals who had allowed open wounds on their hands to be soiled by saliva from the rabid animal. Under these circumstances, exposure could not be ruled out; consequently, they were included with those having received actual bites. Recent cases of human rabies in which exposure was definitely not by bite have been reported in the nation.

COMPLICATIONS

While there were no fatal complications from the immunization procedures, there were 14 who showed reactions of one type or another which were severe enough to warrant cessation of treatment. They ranged from mild generalized symptoms, with low grade fever, to rather severe reactions requiring hospitalization of two individuals.

The most severe complication occurred after the eighth treatment in a young adult who developed local reactions and allergic symptoms resembling a severe "cold" accompanied by frontal headache and low grade fever. This individual apparently had complete subsidence of symptoms on the third day after treatment was stopped. He then started on a cross-country trip but before reaching his destination, he became comatose and had to be taken to the hospital. He showed extreme symptoms of encephalitis with generalized central nervous system involvement. After a rather stormy course of 10 days, he made a complete recovery with no residual effects.

There were no after effects in any of the 14 in which reactions occurred. It is interesting to note that these reactions occurred after the seventh injection and never later than the eleventh. Both the medium and the average showed that the ninth injection was the one which produced reactions necessitating stoppage of treatment.

SUMMARY

An outbreak of rabies in Richmond occurred during July and August, 1951, with 29 animals being diagnosed as positive by laboratory examination, and an additional 5 being suspicious. Quarantine of the area affected was set up, and the outbreak was brought under control with a minimum period of time involved.

Diagnosis of rabies was based on approved laboratory methods. Further follow-up was done by animal inoculation. Cases of distemper did not confuse the diagnosis of rabies. St. Louis viral studies have revealed the possibility of a second entity similar to rabies, but as this is theoretical, no Public

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Health Worker would be justified in failing to report as positive any microscopic examination which showed typical Negri bodies.

During the outbreak, 88 persons were given anti-rabies treatment. Fourteen of these showed some type of reaction.

CONCLUSIONS

During an outbreak of rabies, all laboratory examinations revealing the presence of Negri bodies should continue to be reported as positive. Adequate facilities for quarantine, impounding of stray dogs, vaccination of dogs, and immunization of exposed persons, should be maintained.

Procedures outlined in the Control of Communicable Diseases in Man should be followed as they apply to rabies control.

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Alcohol Retained in Body Determines Intoxication.

The amount of alcohol that a person's body fails to oxidize or eliminate determines the degree of his intoxication, rather than the amount of alcohol that he consumes, a medical consultant stated in the Journal of the American Medical Association. "In other words," the consultant pointed out, "it is the unburned alcohol in the body that causes intoxication.

"The average 150 pound man oxidizes and eliminates about seven to ten cubic centimeters of absolute alcohol per hour. If he would space his drinks, he could drink a pint of 100 proof liquor in 24 hours without showing physical or chemical signs of intoxication." It was stressed, however, that the presence of food in the stomach delays absorption of the alcohol, and the dilution of the alcohol in the stomach is also a factor to be considered.

"The only reliable way to determine a person's state of intoxication is to test the per cent of alcohol in his blood, breath or urine," it was stated. An average person who consumed five alcoholic drinks, each containing one-half ounce (15 cubic centimeters) of absolute alcohol, within three hours would have consumed two and one-half ounces of alcohol (75 cubic centimeters) according to the consultant. This amount could cause a maximal blood alcohol of about 0.12 per cent by weight, if no oxidation took place. However, in the three hours of drinking, the individual would oxidize about one ounce of alcohol, thus reducing the blood alcohol to a maximum of about 0.08 per cent.

"From the standards set by the Committee on the Study of Motor Vehicle Accidents of the American Medical Association and the Committee on Tests for Intoxication of the National Safety Council, this would be interpreted as an amount that is usually associated with some loss of that clearness of intellect and self-control that he would ordinarily possess, but the obvious physical symptoms of the person should be taken into consideration before a diagnosis of intoxication is made," the consultant said.

"The unfortunate part of the calculations based on the amount consumed is that they can show a possible maximum blood alcohol, but the actual amount is usually unpredictably below this figure."

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THE USE OF CATION RESINS IN THE TREATMENT OF EDEMA*†

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The use of ion exchange resins in chemistry and industry has been an accomplished fact for approximately twelve to fifteen years[17,3], but their value in the field of medicine has not been fully explored. A principle allowing the removal of certain ions from solutions of various types has led to speculation and experiment in medicine including the fixation of dietary sodium for alleviation and prevention of edema formation[3,8,9,10,18], the therapy of hyperkalemia in renal failure, the determination of free gastric acidity without intubation, decalcification of blood to inhibit coagulation[3,17], decalcification of bone specimens for sectioning[17] and other situations not yet fully explored. This brief discussion is limited to medical uses of the cation resins and the many, varied and precise maneuvers of chemistry and industry involving the use of ion exchange resins may be found in detail elsewhere.

This report is concerned primarily with the use of cation exchange resins in the treatment and prevention of cardiac edema, edema of cirrhosis of the liver and nephrotic edema. We have had no extensive experience with edema of pregnancy, a matter of some promise in this field.

The control of edema by sodium restriction is accepted, but the accomplishment of satisfactory sodium restriction is not always possible. Therefore, a nontoxic substance that can be ingested for the fixation of dietary sodium has aroused increasing interest since Dock's first suggestion[1]. It has been shown quite clearly that a cation resin may be used for this purpose in patients with edema[4,5,7]. These same authors have pointed out the hazards and limitations of these substances[4,5]. Prior to these clinical studies it had been thoroughly demonstrated that the cation resins used were non-toxic for animals and did not interfere with their growth reproduction hematological picture or life span[6,2].

It is not necessary here to review the theoretical mechanisms by which polyacrylic polycarboxylic cation exchange resins bring about fixation of sodium when taken internally and mixed with the food. It is important to say that these resins are reasonably efficient and a dosage of 45 to 60 grams of Resodec R* for example will fix 40 to 60 meq., of sodium depending upon the magnitude of sodium intake and the degree of congestive failure. It is also important to indicate that cation resins take up potassium magnesium and calcium as well sodium magnesium and calcium fortunately are not fixed in sufficient amounts to raise clinical problems of deficiency and this is also true of vitamins and other minerals. The problem of hypokalemia from potassium fixation was conquered by the proper mixture of the ammonium and potassium forms of cation resins so that as much potassium was absorbed as was fixed. Since this arrangement we have seen no suggestion of hypokalemia.

It is well to indicate in the beginning that the cation resin is not a cure-all, and not a complete solution for the dietary management of congestive heart failure or edema patients. A subsequent paragraph will indicate that the capacity of the resin is not sufficient to take care of a full diet and the patient must not expect to eat as much salt as he wishes, for some degree of dietary sodium restriction will be necessary even with full resin dosage. Furthermore, it is important to state that the cation resin is not indicated in all heart failure patients. Many patients with congestive heart failure respond to rest, digitalis, and moderate restriction of their sodium intake and will remain compensated for months or even years under such a regime. However, a number of patients with heart failure and edema are

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*Resodec R used in the study has been furnished by Smith, Kline and French, of Philadelphia. Resodec R is a highly cross-linked, polyacrylic polycarboxylic cation exchange resin. It contains enough of the potassium resin to provide 20 mm. Eq. of potassium per dose and the balance is made up of ammonium resin. This figures out 3.33 grams (dry weight) of potassium resin and 11.67 grams (dry weight) of ammonium resin[3].

unable to reach a suitable level of sodium restriction. In these patients a proper administration of a cation resin may be extremely helpful as an adjunct in treatment. This is particularly true of patients with long standing chronic congestion who have even become refractory to mercury following the exhibition of the cation resin brilliant results with mercurial diuretics may be restored and longer intervals between injections accomplished. In brief, the more severe and more refractory cases of congestive heart failure appear to be the ones to whom this principle may be properly applied.

In addition, as noted below, it has been our experience that the obstinate edema associated with nephrosis sometimes may be profitably attacked by this method. Particularly striking have been several instances in which considerable diuresis in cirrhosis of the liver with acites has followed the use of the resin alone, whereas potentiation of mercurial diuretics following the use of the cation resin has been brilliant in several instances. Recently, an excessively edematous patient suffering from constrictive pericarditis was better fitted to face the ordeal of operation following the use of the cation resin.

The dosage of Resodec[R] is usually 15 grams in fruit juice after each meal, making a total of 45 grams daily. Sixty grams appears to offer little if any advantage. Since the capacity of a cation resin is limited in this dosage to the fixation of 50 to 100 meq. sodium daily the patient should be on a moderately low sodium diet of 1,000 to 1,200 mgms. of sodium daily. A cation resin must not be expected to fix the large amounts of salt in a regular unrestricted diet. In previous studies[5] we have indicated that the sodium binding effect of the resin continues for some time after resin is discontinued by mouth, generally two or three days. Therefore, administration of the resin four days of each week is usually enough or a smaller daily dose of 15 grams morning and night will generally keep the effect going.

The taste of most cation resins is not pleasant but a minor inconvenience compared to the relief obtained. Constipation, sometimes diarrhea, and rarely vomiting, may occur, but vomiting has only occasionally proved troublesome. Small initial dosage often solves this problem. The question of the acidosis often accompanying resin administration is discussed below.

The effects of the administration of an ammonium and potassium resin (Resodec[R]) may be briefly discussed. It is not always possible to explain all effects or to evaluate the therapeutic value of the resin while other measures of treatment are being used. However, it was our impression that the resin produced a decided restriction of sodium re-absorption in the three non-edematous cases studied, perhaps a mild acidosis, and perhaps a slight elevation of plasma chloride. As noted above, the effect lasted several days after the resin was discontinued.

In cardiovascular patients with edema, twenty of twenty-eight patients showed added clinical improvement following the use of the resin which appeared to be attributable to the resin. In eight of these the results were quite striking, as conventional measures with digitalis, ammonium chloride and mercury had failed until the resin alone.

In fifteen of twenty-six patients marked potentiation of a mercurial diuretic occurred following the use of resin, with a slight or indifferent effect in the remaining eleven. The remaining congestive heart failure patients did not require a mercury diuretic. In diuresis careful and specific trials with ammonium chloride had previously failed. Other observers have noted this but have failed to elucidate the true reason for the apparent superiority of an ammonium potassium cation resin over the equivalent amount of ammonium chloride.

The whole question of potentiation of mercurial diuretic by a cation resin needs further study. In this regard it may be noted that Resodec[R] brought about an elevation of the plasma choloride from a lower to a higher figure in nineteen out of twenty-two patients with congestive heart failure where this observation was made. Furthermore, a mild to moderate acidosis was produced in twelve out of twenty patients with congestive heart failure. Cation resin administration, therefore, leads to the two states commonly accepted as necessary for potentiation of mercurial diuresis—namely, adequate plasma chloride levels, and diminished plasma bicarbonate levels[12,13]. Again, it is not clear whether these two effects are the result of ammonium chloride absorption associated with administration of ammonium-potassium cation resin or a combination of this and the restriction of sodium absorption.

At no time have we encountered an uncompensated acidosis with hyperpnea. The lowering of the plasma

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bicarbonate has been temporary, as noted in Fig. 1, and the effect of the acidosis has been in general similar to the familiar effects of ammonium chloride. The potassium form of the resin mixed with the ammonium form in proper proportion offsets to an appreciable extent the loss of fixed base and partially prevents acidosis[19]. We have had only limited experience with the anion-cation mixture[14].

The favorable effects of restriction of sodium absorption with temporary acidosis is well illustrated in Mrs. H. E. (Fig. 1). This lady of sixty with

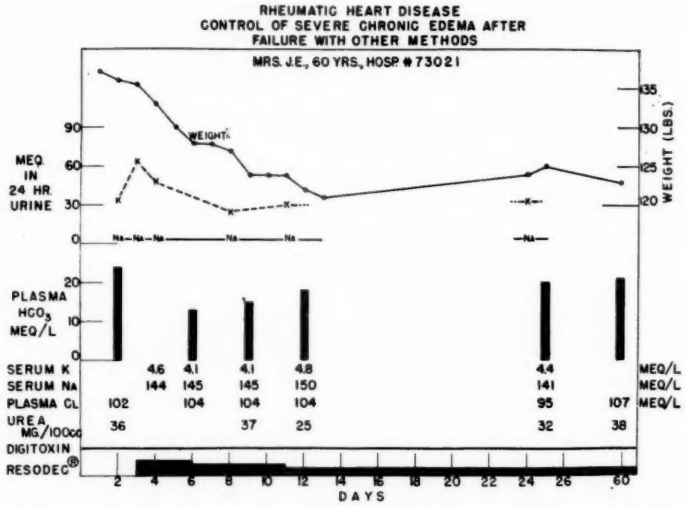

Fig. 1.—Mrs. H. E.—Rheumatic heart disease with aortic stenosis and regurgitation and mitral stenosis and regurgitation. Chronic congestive heart failure with edema requiring ammonium chloride and mercurial diuretic every ten days for all year prior to resin administration. Maintenance digitalization with digitoxin unchanged. Hospitalization first few days, outpatient thereafter.

Table indicates mitral fall of plasma bicarbonate following resin administration with gradual recovery to near normal levels on continued resin dosage. Mercu y unnecessary after resin was started.

advanced rheumatic heart disease, aortic regurgitation and mitral stenosis had suffered from congestive heart failure and edema for months. For over a year she had received a mercurial diuretic every ten days, each injection preceded for three days by six to eight grams of ammonium chloride daily. This routine became less and less effective and she entered the hospital for ammonium potassium cation resin therapy on day "one" of Fig. 1. Up to this time she had been on a diet of 1,000 mgm. of sodium to which she had faithfully adhered. Complete digitalization with digitoxin had been maintained for years and the daily ration unchanged. No medication was given except the usual digitoxin and Resodec[R], although excreting no urinary sodium remained at zero following Resodec[R] in spite of steady diuresis and loss of edema. Urinary potassium excretion was maintained at adequate levels and serum potassium remained normal. Plasma chloride levels remained normal in spite of marked diuresis and a mild to moderate loss of plasma bicarbonate returned toward normal even with continued resin administration. The patient was discharged on the fifth hospital day and handled thereafter as an outpatient. On the twelth day Resodec[R] was reduced to thirty grams daily and clinical comfort and relatively edema free weight maintained on this dosage. No diuretic of any kind has been given since the first day of Resodec[R] and she has continued to remain up and around with minimal edema on this regime for nearly five months. The period of observation has now extended to one hundred and fifty days on resin instead of the period of sixty days shown in Fig. 1.

The foregoing does not imply that cation resin administration is free from danger. Two major hazards exist, the low salt syndrome[15] even in the presence of relatively good renal function and the aggravation of an already existing kidney inadequacy.

The low salt syndrome has occurred twice with

us during resin administration and fortunately this state has been corrected without mishap on both occasions. The best safeguard against this hazard is a thorough appreciation of the syndrome and the attendant dangers.

Occasionally it may be necessary to use the cation resin in the face of renal insufficiency, but the danger is considerable and, as in all such situations, edema loss is generally bought at the price of azotemia.

In one patient with chronic glomerular nephritis, uremia, and massive edema, the resin was deliberately used in the face of renal insufficiency with a resulting moderately severe acidosis. Considerable edema fluid was lost, with relief to the patient, but the blood urea mounted rapidly from 88 mgm./100 cc. to 177 mgm./100 cc. Renal insufficiency has been commonly accepted as a prime contraindication to cation resin administration. Effects of sodium depletion by resin therapy during uremia and the unfavorable mechanisms involved have been recorded[16]. Six patients with cirrhosis of the liver, edema and ascites have received the resin from four to fifteen days. Every one of the six patients studied had a proved biopsy diagnosis of portal cirrhosis. Two patients showed a marked diuresis on resin alone. The remaining four showed a marked mercurial effect following resin, although mercury and ammonium chloride had previously failed in two.

Four patients with the nephrotic syndrome of chronic nephritis were studied. One appeared to be a failure largely because of the accompanying glomerulo-nephritis; obviously he was not a suitable candidate for the cation resin which produced nausea. In another who had the nephrotic syndrome associated with chronic glomerulo-nephritis, the renal function was good at the time, and a moderate diuretic response followed the cation resin alone. Also marked mercurial diuresis helped bring about a loss of 42 pounds in a brief period of 23 days. Here, again, the diuresis may have been spontaneous and coincident with resin administration, but at least the patient had been refractory to the usual methods of treatment for six weeks prior to resin administration. This patient has remained well and edema free for several months with complete resumption of all activity. In the two remaining nephrotic patients the results of resin therapy were poor. Renal failure increased and azotemia mounted so that the resin was discontinued. Again, it should be emphasized that adequate renal function in the nephrotic syndrome should be prerequisite to cation resin administration.

Two patients with constrictive pericarditis were observed following resin administration. One appeared to show a moderate loss of edema from resin effect while the other benefited decidedly by moderate diuresis from the resin alone, and moderate to marked potentiation of a mercurial diuretic.

The question has arisen on a number of occasions as to just what simple rules should be followed to ensure relative safety during administration of a cation resin. Obviously, all physicians may not be able to follow patients with detailed chemical analyses.

It would seem unwise to administer resin to a patient with an appreciable degree of renal insufficiency and, generally speaking, nitrogen retention in the blood should be considered a contrainindication to its use. Daily weight recordings are essential to the management of an edematous patient. Finally, a reasonable good appetite and a reasonable good flow of urine should be present before a cation resin administration. Moderate to marked diuresis nearly always follows a mercurial diuretic and failure of mercurial diuresis during the course of resin administration may well be indicative of renal failure, or the low salt syndrome, or both. Hyperpnea, nausea and vomiting are signals for immediate discontinuance of the resin, and these symptoms should be followed up by determinations of plasma cloride, plasma bicarbonate and blood urea or similar blood nitrogen studies. The therapeutic use of a cation resin is not without danger and requires caution and detailed attention.

SUMMARY

An ammonium-potassium cation resin has been given to three subjects without edema and forty patients with edema. Suitable clinical and laboratory studies have been carried out on these individuals.

This cation resin restricts sodium absorption in therapeutic dosage to the order of 40 to 60 meq., of sodium daily. Sodium dietary restriction is necessary to a moderate degree during resin administration.

The low salt syndrome and aggravation of existing renal insufficiency are the chief hazards from cation resin administration. Failure to recognize these

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dangers may lead to serious states, including uremia.

Cation resin therapy may be a helpful adjunct in selected cases of heart failure with edema and cirrhosis of the liver with ascites and edema. It is not a cure-all and should not be used in all patients with edema.

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PRIMARY CARCINOMA OF THE LIVER†

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Virchow is credited with the dictum that "such organs as are frequent sites of metastatic tumor are rarely the site of original neoplasm". This is particularly true when applied to malignancies of the liver, for, while the liver is frequently invaded by metastatic tumor, the incidence of primary carcinoma of the liver is rare, indeed. Nevertheless, it occurs with sufficient frequency to be of importance, especially in the differential diagnosis of obscure abdominal disease, and is rarely diagnosed antemortem. This paper analyzes thirty-eight cases collected at the Medical College of Virginia Hospitals, the McGuire Veterans Hospital, St. Lukes, St. Elizabeths, and Stuart Circle Hospitals.

INCIDENCE

The percentage of cancers of the liver that are primary varies from 1.2 per cent to 17 per cent as reported by various authors quoted by Warvi.[1] The overall autopsy incidence of primary carcinoma of the liver averages about 0.5 per cent in the western world, as reported by Charache[2]. The total number of cases recorded in the world medical literature is now well over 1400.

The age distribution of primary carcinoma of the liver ranges from that of a case reported in a fetus[3] to very old age. It is very definitely a disease of the sixth and seventh decades, however. In our series there was one case occurring in each decade to age 30, three from 30 to 39, 26 from 40 to 70, three from 70 to 79, and two in the 80 to 90 age range.

There is a male preponderance reported in the literature. Our cases showed about a two to one ratio, consisting of 25 males and 13 females. There were seven colored cases and thirty-one white cases, which is probably roughly parallel to the admission rates of the two races. Gustafson,[4] in reviewing 24,000 autopsies in Bellevue Hospital over a period of thirty years, found no increased incidence in negroes but a definitely increased incidence among Chinese and Japanese.

PATHOLOGY

Primary carcinoma of the liver may be divided into three major groups: hepatocellular carcinoma, or hepatoma; cholangioma, and cholangiohepatoma.

Hepatoma arises from parenchymatous liver cells. It is frequently associated with cirrhosis, either as multiple nodules or in the form of a massive tumor growth which may involve a large area of the liver, but, except for grouping of secondary growths around it, may leave the rest of the liver relatively normal. The microscopic picture is one of liver cells arranged in irregular fashion, showing great variation in cell type, abnormal staining features, mitoses, and multinucleated and giant cells.

The second large group morphologically is cholangioma, a neoplasm arising from intra-hepatic bile duct cells. The massive solitary form of tumor is not seen, the growth being scattered throughout the liver. Microscopically it is composed of columnar or cubical cells which may be arranged in duct-like structures.

The third group consists of cholangiohepatoma which is composed of neoplastic proliferation of bile duct cells and liver parenchymal cells. There is usually a predominance of the bile duct character, for this tends to grow and spread more rapidly.

Hepatomas outnumber cholangiomas from a three to one ratio, according to Hoyne,[5] to 92 per cent of 49 cases reported by Wilbur.[6] In our series there were 26 cases of hepatoma, 11 of cholangioma, and 1 of cholangiohepatoma. Twenty-eight cases were diagnosed by autopsy, ten by biopsy. Portal cirrhosis was present in 11 of 26 cases of hepatoma. Biliary cirrhosis was present in 1 of 11 cases of cholangioma. These do not approach the figures of Ewing,[7] who believed that cirrhosis was associated with 85 per cent of the cases of hepatoma and 50 per cent of cases of cholangioma, but are in line with figures given by others.[8,1]

The liver was reported to be large, or above 2000 gms., in 20 of the 23 cases of hepatoma in which the

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size of the liver was noted, and in six of the seven cases of cholangioma in which the liver size was noted. The liver in the case of cholangiohepatoma was 1500 gms. The largest weights given were 4380 gms. for a hepatomatous liver, and 4325 gms. for a cholangiomatous liver. Nodules were noted to be numerous or scattered throughout the liver in all except two cases of hepatoma which had single tumors. One of these was quite small and was found by chance in an autopsy of a patient who died with chronic leukemia. The other was quite large and was diagnosed by biopsy at the time of an exploratory laparotomy.

Ascites was reported as being present in four cases of cholangioma, twelve cases of hepatoma, and in the case of cholangiohepatoma. In one case of hepatoma and one case of cholangioma the fluid was noted to be bloody. The spleen was not enlarged unless there was portal obstruction.

Intra-hepatic metastases are common and probably account for the extensive nodule formation throughout the liver. The spread may be by lymph channels, but is largely hematogenous since the cancer cells are prone to invade blood vessels and may form thrombotic masses is hepatic veins, often extending into the portal or more distant veins. This occurred in six cases. Gregory[9] reported a case of tumor thrombosis of the inferior vena cava and right auricle with symptoms of vena caval obstruction, and found eleven other cases in the literature.

Extra-hepatic metastases are surprisingly uncommon, occurring in 15 of 27 autopsied cases on which pertinent data was available. Two of these were tumor thromboses of the protal vein. There were metastases to regional lymph nodes seven times, to one or both lungs six times, to the pancreas three times, to the adrenals twice, to the stomach twice, in one of which the entire anterior wall was replaced by malignant cells with ulceration, to the vertebral bodies twice, to the skull and ribs once, and to the brain, kidney, heart, and pericardium once. Thus there were 28 metastases in 15 cases which showed extra-hepatic metastases. Metastases have also been reported to the pleura, long bones, epidural space, spinal cord, peritoneum, and prostate. Auerbach[10] reported a case with extensive skeletal metastases and panmyelophthisis. One of our cases that showed a profound anemia with hemoglobin of 58 per cent and RBC 2.8 had metastases to the vertebral bone marrow. There were extrahepatic metastases in the case of the cholangiohepatoma. It would appear that cholangiomas are more invasive, for extra-hepatic metastases occurred in 4 of 5 cases of cholangioma and 10 of 21 cases of hepatoma.

ETIOLOGY

The variations in incidence of primary carcinoma of the liver are arresting and undoubtedly contain clues as to its etiology. Bonne[11] reported primary hepatic carcinomas to be the most frequently encountered malignancy in the Netherland East Indies and Singapore, and the second most frequently encountered malignancy in Japan. This has been attributed by Strong[12] and others to the high incidence of intestinal parasites and schistosomiasis in these areas with chronic irritation and development of cirrhosis. An azo dye used widely as a coloring agent in the Japanese diet induces primary carcinoma of the liver in rats and mice.[13] Webb[14] considers dietary deficiencies to be the common denominator explaining the racial and geographic distributions. Symmers[15] reviewed five cases of schistosoma hematobrium involvement of the liver in which cirrhosis was found and concluded that the cirrhotic changes were most likely attributable to the miserable dietary conditions under which those patients lived. Experimentally, avitaminosis B has been shown to predispose to carcinoma in the presence of excessive dietary fats or administration of carcinogenic agents.

As has been stated, there is a close relationship between cirrhosis and primary carcinoma of the liver. Loesch[16] has estimated that on the average one out of eight cases of cirrhosis develop hepatoma.

CLINICAL COURSE

The presenting signs and symptoms of primary carcinoma of the liver are not unlike those of malignancy in other parts of the body; i.e., loss of weight, loss of strength, malaise, anorexia, etc., plus the particular indications which locate it in the liver.

Pain was the most prominent symptom in our series, being present in 25 of 36 patients (69 per cent). It was present in every case that had extra-hepatic metastases and was present more uniformly in cholangioma (8 of 11 cases) than in hepatoma (16 of 24 cases). It was the presenting complaint alone or in combination with other symptoms in 21 of 36 cases (58 per cent). It occurred in varying severity from agonizing to a mild discomfort. It usually

occurred in the right upper quadrant, epigastric or umbilical regions but was most severe in the back in several patients.

Weight loss varying from slight to 50 lbs. was present in 23 patients (64 per cent). Weakness and malaise were present in 18 patients (50 per cent) and was marked in 7.

Gastro-intestinal symptoms were frequently present but followed no characteristic pattern. Appetite was specified as being poor in 11 cases, indigestion, "gas", belching, or nausea were present in 14, and change in bowel habits occurred in 8 patients, 3 of whom had diarrhea and 5 constipation.

The most frequently noted physical finding was an upper abdominal mass which occurred in 26 of 37 cases (70 per cent). Ascites was noted to be present clinically in 15 cases (40 per cent). It was bloody in only 2 cases.

Edema was present in the case of cholangiohepatoma, in 6 cases of hepatoma, and 2 cases of cholangioma. Fever was present at sometime in the duration of 20 cases (54 per cent), especially terminally. Anemia of less than 12 gms. was present in 19 cases (50 per cent). The white count was very variable, and when elevated appeared to be related to an intercurrent infection or hemorrhage rather than to the carcinoma. No correlation could be made between the carcinoma and urinary findings or blood chemistries.

Liver function studies were frequently not done and usually were of little value since they were either negative or there was other evidence of severe liver disease when they were positive. Nevertheless, in several cases abnormal liver function tests were the first indication that the liver was the site of the disease process.

X-ray studies may be helpful in making the diagnosis by demonstrating an elevated fixed right diaphragm, a large upper abdominal soft tissue mass, or displacement of stomach or colon by extrinsic pressure. In ten cases one or more of the above abnormalities were demonstrated but x-ray changes are usually a late finding.

The clinical findings in order of frequency were upper abdominal mass (70 per cent), pain (69 per cent), weight loss (64 per cent), fever (54 per cent), jaundice (52 per cent), weakness and malaise (50 per cent), anemia (50 per cent), ascites (40 per cent), gastro-intestinal symptoms of anorexia, indigestion, and change in bowel habits, edema, x-ray abnormalities, and abnormal liver function tests. Similar findings are listed by all authors in case reports but the percentages vary widely, probably depending upon associated or underlying conditions such as the presence or absence of dietary deficiencies, cirrhosis, schistosomiasis, etc. There seemed to be little difference in symptoms on a percentage basis between hepatoma and cholangioma, although this is admittedly a small series.

This is one of the most rapidly fatal of malignancies. The average interval between the onset of symptoms and death was four months, the shortest was two days, and the longest eighteen months. Admittedly the time of onset of symptoms due to carcinoma of the liver was hard to determine in some cases of cirrhosis who had had known liver damage for several years.

DIAGNOSIS

The diagnosis of primary carcinoma of the liver was suspected in only two cases in this series, which indicates that is it not considered in differential diagnosis as frequently as it should be even though it is a diagnosis that can be finally established only by biopsy or autopsy.

Our cases fit well into the four classical groups suggested by Ewing[7]: (1) Those in which no symptoms are detected and the patient dies suddenly from hemorrhge or after an illness of several days. (2) Those in which latent carcinoma is found among patients who succumb to cirrhosis or other diseases. (3) Those in which the usual history of cirrhosis terminates rapidly with hepatic tumor, jaundice, ascites and cachexia, and (4) those in which the usual history of a malignant tumor, indicating from the first involvement of the liver, develops among previously healthy patients.

Groups one and two admittedly cannot be suspected clinically, but it is important that three and four should be diagnosed more frequently and earlier if the patient is to have a chance for effective treatment. It is our feeling that this disease can be diagnosed clinically if the signs, symptoms, and clinical course are kept in mind.

TREATMENT

Treatment of primary carcinoma of the liver is entirely surgical, since x-ray and other agents are of no value. We are unable to agree with those who

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believe that surgical exploration is not justified as a diagnostic procedure and therapeutically offers nothing in primary carcinoma of the liver or in those diseases most apt to be confused with it, such as cirrhosis of the liver.

Most patients in whom this disease is suspected have a hopeless outlook. Their one chance for survival is that they might have a non-malignant condition amenable to surgery, or that they might have a single hepatoma nodule that can be removed. These patients will largely fall in group four, and it is our opinion that patients with that clinical picture should have the benefit of an exploratory laparotomy. We are unable to support this statement by the course of any patient in our series, but Wallace[17] collected 29 cases of hepatomas treated by resection, of which five lived two years, three lived three years, and four lived for more than five years.

SUMMARY

The hypothetical patient with primary carcinoma of the liver will be a male in his 60's with a three to six month history of indigestion, dyspepsia, and gradually developing upper abdominal pain and fever who finally presents himself to his physician probably because of pain, upper abdominal mass, weight loss, or jaundice. He has lost some weight, possibly a considerable amount, and his appetite has diminished. His abdomen has become full and distended. On physical examination he shows obvious wasting, usually jaundice, and frequently evidence of ascites is present. As a rule his liver is easily palpated and nodules may be felt. Laboratory studies may show liver damage, anemia, and leucocytosis. He fails to respond to transfusions, high protein diet, and other supplementary measures and finally dies some three to ten months after his symptoms began.

The literature on primary carcinoma of the liver has been reviewed and thirty-eight cases have been analyzed.

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DISCUSSION

DR. ROBERT F. BONDURANT, Roanoke: We have just heard a good paper on a little discussed subject. In round figures there have been only 1,600 cases of primary carcinoma of the liver reported in the literature. Here we have 38 well analyzed cases. Dr. Higgins and Dr. Thompson are to be congratulated for this contribution.

As has been pointed out, there is a puzzling variation in the geographic incidence of this disease. In the Orient it accounts for 14% of all carcinoma, whereas in the United States and in Europe it accounts for only 1% to 2% of all carcinoma.

Any process that can cause destruction or inflammation of liver tissue may be considered potentially carcinogenic. Two-thirds of the patients with primary carcinoma of the liver have an associated cirrhosis. Anywhere from 4% to 8% of patients with cirrhosis develop primary carcinoma of the liver. These facts cannot be taken at face value however. There has been an increase in the incidence of cirrhosis in the past few years and no commensurate increase in primary hepatic carcinoma. It is well known that cirrhosis plays no part in this disease in infants and children.

Hemachromatosis, a rare disease in itself, is often complicated by primary carcinoma of the liver. The incidence is about 10%.

There have been many suggestions made to explain the variation of the incidence of primary hepatic carcinoma geographicaly.

(a) Parasitic Infestation of the Intestinal Tract.—There is much evidence to suggest this, but the populace in the Nile Valley have a high degree of parasitic infestation without an equally high incidence of primary carcinoma of the liver.

(b) Some have suggested it is a disease of the pigmented races. The Bellevue Hospital statistics about Negroes just quoted in this paper would not support this.

(c) Probably the most readily acceptable theory is that of dietary deficiency.

Dr. Higgins and Dr. Thompson have explored the clinical features of this disease thoroughly. The key to this diagnosis is a high index of suspicion. This is especially

true in patients with cirrhosis and in patients with hemachromatosis.

This paper advocates surgical exploration and ample reason for this advocacy is given. Some would suggest diagnosis by needle biopsy or by peritoneoscopic examination. Ruddock, in 1939, and a group at the Mayo Clinic in 1950 report in combination 700 to 800 cases of tumors of the liver. They were able to accurately diagnose 93% to 94% of them by peritoneoscopic examination.

The diagnosis of primary carcinoma of the liver rests on direct inspection of the liver and, at times, biopsy. A word of caution: whenever biopsy is to be done the prothrombin value should be known. Many of the fatalities occurring after biopsy have been associated with low prothrombin values.

It is important to differentiate the primary disease from metastatic malignancy of the liver. In metastatic malignancy the course of the disease is longer, splenomegaly is seldom prominent, and cirrhosis is usually absent. The primary site is usually found by ordinary means of diagnosis.

In differentiating the patients with cirrhosis complicated by malignancy from those with cirrhosis alone, pain is probably the best clinical guide.

Primary carcinoma of the liver may at times be confused with almost any condition in the abdomen from infection to the surgical emergencies.

As surgery early in the disease is the only definitive treatment, early diagnosis is important. Early diagnosis is possible by the means suggested in this paper.

I wish to thank Dr. Higgins and Dr. Thompson for the privilege of discussing this paper.

Possibility of Cure Best in Early Diagnosis of Cancer Larynx.

Early diagnosis and treatment of cancer of the larynx can result in a high percentage of cures and restoration of the voice, according to an article in the May J.A.M.A. The location of the larynx and the type of malignant lesion that occurs in it make it possible to cure a larger percentage of cancers in this organ than in any other part of the body except the skin, according to Dr. Gabriel Tucker, of the department of bronchology, esophagology and laryngeal surgery, graduate school of medicine, University of Pennsylvania, Philadelphia. "Therefore," he added, "it is essential that effort is made in all suspicious cases to secure early diagnosis by careful examination and careful study of the symptoms. These early symptoms are hoarseness and local discomfort."

Surgical removal, irradiation, or, in certain cases, both, are the well-established methods of treating this disease, Dr. Tucker stated. Surgery is the treatment of choice in selected cases. Irradiation has proved most valuable in cancer in the older age group and in those patients whose general condition makes surgery an unwise procedure. The use of antibiotics in surgical cases and in surgical treatment of postirradiation cases has aided in obtaining favorable results, he said.

Dr. Tucker reported that of a group of 152 patients with laryngeal cancer who were treated with surgical removal of part of the larynx, 86 per cent were free from recurrence of the disease for five years or longer. Of 102 patients treated by complete removal of the voice organ, 61 per cent were free from recurrence for five years or longer.

Although complete results could not be obtained in cases where cancer of the larynx was treated by irradiation, Dr. Tucker said be believed five year cures were obtained in at least 25 per cent of those patients treated by this method.

"Rehabilitation and the possibility of developing an esophageal voice following the complete removal of the larynx are excellent," Dr. Tucker pointed out. "In the older age groups, difficulty with the esophageal voice may be experienced, and many patients prefer using an artificial larynx (electrolarynx), which produces a good voice and enables the patient to resume his place in the social and business world."

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THE MANAGEMENT OF CHRONIC COR PULMONALE WITH FAILURE*

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DEFINITION

Chronic cor pulmonale or pulmonary heart disease, by definition is heart disease resulting from pulmonary disease; or, more specifically, right ventricular hypertrophy with or without heart failure, resulting from hypertension of the pulmonary vascular system. Cyanosis and polycythemia are usually associated.

UNDERLYING PULMONARY DISEASE

The basic pathology in cor pulmonale is usually found in the lungs, and diseases such as emphysema, diffuse fibrosis, silicosis, Boeck's sarcoid, berylliosis, tuberculosis, bronchiectasis, kypho-scoliosis and multiple emboli constitute the majority of basic conditions found. However, a condition such as an aneurysm of the ascending aorta, causing pressure on the pulmonary artery and restriction of pulmonary artery flow, will lead to right ventricular hypertrophy and subsequent failure. Emphysema, though, is the most frequent offender, as indicated by a recent report[1] that emphysema was present in 69% of 48 patients with chronic lung disease with circulatory complications.

PATHOLOGICAL PHYSIOLOGY

Before discussing the management of cor pulmonale with failure, it appears important to review the pathological physiology of the underlying pulmonary disease. Emphysema will thus be of primary concern, whether it be the predominant factor as with obstructive emphysema, or whether it be a compensatory type as with silicosis, tuberculosis and kyphoscoliosis, providing a mechanical factor such as an aneurysm of the ascending aorta is not present.

The pathological physiology of emphysema will be divided into pulmonary features and circulatory features.

PULMONARY FEATURES: The lungs characteristically present a loss or fragmentation of normal pulmonary elastic tissue, a narrowing of the bronchial tree, due to bronchiolar spasm, secretions, or thickening of the mucosal lining, and numerous blebs, bullae and air cysts. They then become hyperinflated, as demonstrated by physical examination and x-ray, since the alveoli are markedly widened due to the breakdown of elastic tissue. There follows an increase in total lung capacity, but, more important, an increase in the residual volume. This latter is usually associated with an increased total lung capacity; however, the total *functional* lung capacity may be reduced because of air trapped in the blebs, bullae or air cysts which do not communicate with the trachea. Vital capacity which is a function of total lung capacity, and residual volume may be either normal or reduced, the latter occurring as the residual volume becomes greater.

Maximum breathing capacity is usually reduced out of proportion to the decreased vital capacity, and with this there is a decreased breathing reserve[2] and dyspnea.

In addition to the effects of emphysema on the mechanics of breathing and lung volume, there is an alteration of the process of gas exchange.

The exchange of oxygen and carbon dioxide between mixed venous blood, and the atmosphere is the major function of the lungs and a decreased arterial oxygen saturation or an increased partial pressure of carbon dioxide is direct evidence of a defective gas exchange. These findings are manifest clinically by hyperventilation.

Defective gas exchange has been found[2,3] to occur in emphysema because of uneven distribution of inhaled air and of pulmonary capillary blood flow. Inhaled air is unevenly distributed because of the bronchiolar narrowing, local variations in elasticity and the presence of air cysts and blebs which trap air, as mentioned above. This leads to under-ventilation of some alveoli and over-ventilation of others. The normal ventilation of alveoli with reduced capillary blood flow results in the formation of an increase in the respiratory dead space since the transfer

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*Read before the annual meeting of The Medical Society of Virginia, at Virginia Beach, October 7-11, 1951.

of oxygen and carbon dioxide cannot occur in those areas. This leads to hyper-ventilation of the whole lung, overwork, and then dyspnea. On the other hand, normal circulation to alveoli with poor aeration is in essence a right to left shunt of blood, with resulting anoxemia. If hyper-ventilation of remaining well aerated and well perfused alveoli is adequate, carbon dioxide retention does not occur, but if anoxemia is present, it cannot be corrected significantly by hyper-ventilation since the oxygen saturation of the blood flowing through the alveoli cannot be raised more than 1-2% by inhalation of the 21% oxygen in the air.

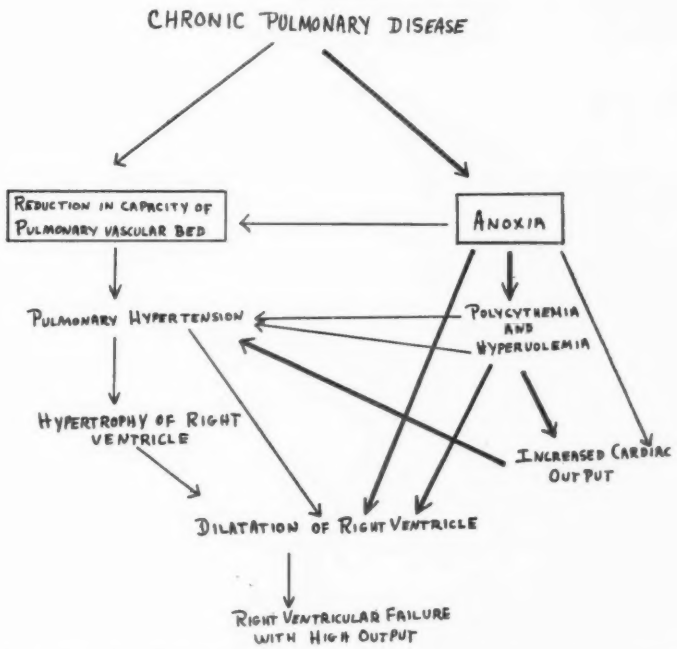
CIRCULATORY FEATURES: Normally, the pulmonary vascular bed exhibits a marked distensibility and it can accommodate at least a three fold increase in pulmonary blood flow before an increase in pulmonary artery pressure[4] occurs. In emphysema, however, due to the structural changes which occur, the vascular bed is restricted. Pulmonary artery hypertension is then favored with any increase in blood flow, such as would occur with exercise or infection. In addition, hyper-ventilation is not adequate, with the increased demand imposed, and anoxia occurs. Anoxia, alone, has been shown to produce pulmonary hypertension, probably through the mechanism of local vaso-constriction[5]; thus there are two factors responsible for pulmonary hypertension in emphysema. Anoxia, which can be corrected with treatment, as will be discussed, is the most important factor in the production of the pulmonary hypertension.

Anoxia, in addition to its effect on pulmonary artery pressure, leads to polycythemia and hypervolemia. Furthermore, it has been shown[6] that cardiac output increases in proportion to the severity of arterial anoxia.

Thus, summing up the effects of anoxia on the circulation, arteriolar constriction, pulmonary hypertension, polycythemia, hypervolemia, and increased cardiac output occur. How these factors are related in the production of cor pulmonale is shown in Fig. I and II.

When the nature of the pulmonary disease is such that anoxia assumes more significance than a restricted vascular bed, as occurs in emphysema, the changes in circulation occur as mentioned, and high output failure results. This is shown in Figure I.*

FIGURE I



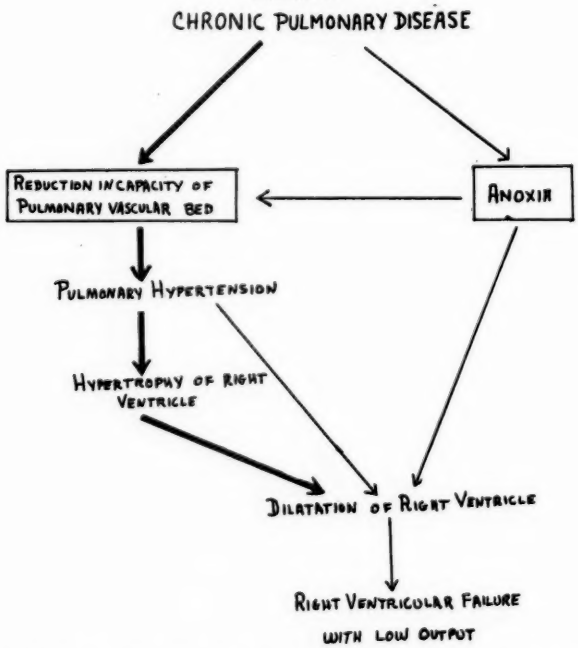
*From "Some Aspects of the Pulmonary Circulation in Normal Man and in Chronic Cardio-Pulmonary Disease," by Andre Cournaud, *Circulation*, 11:641, 1950.

Anoxia, assuming more importance than a structurally restricted vascular bed, as indicated by the heavy arrow, leads to polycythemia and hypervolemia. These in turn lead to an increased cardiac output, which is also affected by anoxia, and this increased output, through a restricted vascular bed, raises pulmonary artery pressure. Anoxia, however, further reduces the capacity of the pulmonary vascular bed by the mechanism of vasoconstriction. Because of the increased output, pulmonary hypertension, increased blood volume and direct effect of anoxia on the strained right ventricle, the myocardial fibers reach their maximum stretch, and right ventricular failure occurs.

Heart failure, in this instance, can likely be reversed if anoxia is corrected.

When the nature of the pulmonary disease is such that the vascular bed is severely restricted, and assumes more significance than anoxia in the production of cor pulmonale, as occurs in silicosis, heart failure is of low output type. This is shown in Figure II.*

FIGURE II



In this instance the restriction of the vascular bed is of most importance, as indicated by the thickness of the arrows, and another train of events leads to heart failure. Since the pulmonary structural changes cannot be reversed, the heart failure is for the most part irreversible.

STUDIES TO BE DONE

Before treating chronic cor pulmonale with failure, it is important that certain studies be done, which will be helpful in establishing the diagnosis as well as in establishing a base-line by which the effects of treatment may be followed. These are outlined in Figure III.

FIGURE III

STUDIES

X-ray and Fluoroscopy
Electrocardiogram with particular reference to right precordial leads
Venous pressure
Circulation time
Vital Capacity
  a. before and after bronchial dilators
  b. before and after digitalization
  c. as a means of following progression of pulmonary disease
Hematocrit

(1) X-rays include a chest plate and chest fluoroscopy. The underlying pulmonary disease is usually apparent, in addition to which the presence

of a prominent pulmonary conus is evidence of pulmonary hypertension.

(2) Electrocardiograms may or may not show evidence of right ventricular hypertrophy. Figure IV shows the electro-cardiographic changes which must be present before a positive or a presumptive diagnosis of right ventricular hypertrophy can be made on an electrocardiographic basis.

FIGURE IV[7]

ELECTROCARDIOGRAPHIC CHANGES

Patterns indicating right ventricular hypertrophy

I. 1. Ratio R to S greater than 1 in V3R and V1.
2. Ratio R to S less in V5 and V6 than in V3R and V1.
3. Delay in onset of intrinsicoid deflection in leads taken over the right side of the precordium.
Normal 0.01 to 0.023 seconds
Right ventricular hypertrophy 0.03 to 0.05 seconds
4. Notching of R in V1 absent except when Q is present.
5. Total QRS duration is less than 0.12 seconds

II. Characteristic changes in V3R.
No changes in V1 to V6.

III. Presumptive evidence of right ventricular hypertrophy.
Prominent S in Leads V4, V5 and V6 with normal V3R; V1 and V2. Increased R wave in AVR.

(3) Venous pressures, done by the direct method[8] and using the anticubital vein at the level 10 centimenters anterior to the skin of the back, may or may not be elevated with right heart failure. Repeated determinations are a guide as to the effect of treatment.

(4) Circulation times, done by the arm to tongue method, are usually prolonged prior to treatment but, after treatment has been initiated, a decrease is indicative of a favorable response.

(5) The vital capacity, which can be done by either the flowmeter, the bellows or spirometer type apparatus, is usually reduced. Here, again, repeated determinations of vital capacity during treatment give information as to the response to treatment.

(6) The hematocrit determination which gives information as to red cell volume, is usually elevated. It is used as a guide in determining the necessity of phlebotomies, as will be discussed.

MANAGEMENT OF COR PULMONALE

The management of cor pulmonale with failure is a challenge. It necessarily includes the treatment of the pulmonary disease and its predisposing factors, as well as the treatment of the circulatory failure. The factors responsible for the production of cor pulmonale with failure in themselves constitute a vicious cycle, and this cycle must be interrupted before treatment is effective. Restriction of the vascular bed, resulting from chronic pulmonary disease, is, as mentioned previously, due to structural changes, and is irreversible; but anoxia, when an important factor, can be alleviated with proper therapy. The improvement of respiratory function and relief of anoxia will be considered under pulmonary phase of treatment.

THE PULMONARY PHASE includes, first, the control of allergic factors. These should be sought and alleviated, especially when asthma, a most frequent offender in emphysema, is present.

Specific medications to relieve bronchospasm and bronchial secretions are of utmost importance in the correction of anoxia. These include such drugs as aminophyllin, adrenalin, ephedrine and isuprel; the latter three may be used as aerosols. Potassium iodide is of importance in loosening bronchial secretions, and thus facilitating their expectoration. Antihistamines, by temporarily controlling the allergic factors, may help relieve bronchospasm and mucosal edema.

Antibiotics are useful in the treatment of pulmonary infections, which are often present and may increase the degree of anoxia so that failure is precipitated or aggravated.

Oxygen therapy, of course, is of utmost and immediate importance in relieving anoxia. Its use is not without danger[3], however, and caution should be exercised during its administration. When patients with cor pulmonale have long standing high levels of arterial carbon dioxide the medullary respiratory center becomes insensitive as a controlling center of respiration, and the carotid and aortic bodies in the neck then assume the important role in control of respiration, by their response to anoxia. If anoxia is then relieved, hypoventilation and finally carbon dioxide narcosis result, since the stimulus to respiration is removed.

There have been reports[9,10] of the use of Cortisone and ACTH in chronic pulmonary disease with improvement as judged by respiratory function studies and X-rays. In addition, these drugs have been used with varying results in *status asthmaticus*. Their use should be kept in mind in the treatment of the pulmonary aspect of cor pulmonale with

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failure, particularly when other measures fail; however, extreme caution must be exercised, since they may aggravate the existing heart failure.

THE CIRCULATORY PHASE of treatment is the management of right ventricular failure. This, briefly, includes digitalization, which has been shown[11] to improve directly the failing ventricle, sodium restriction, use of cation exchange resins and mercurial diuretics. Phlebotomies are of importance in reducing the increased blood volume, and their use should be governed by hematocrit determinations.

It should be re-emphasized that the circulatory alterations in cor pulmonale in emphysema are, for the most part reversible, and since emphysema is present in the majority of instances of cor pulmonale with failure, this fact should offer a great impetus to therapy.

SUMMARY OF CASES

The following tables include a summary of important data compiled in following twenty-one patients with cor pulmonale with failure.

TABLE I

Male	**18**
Female	**3**
Total	**21**
Ages	
Oldest	76
Youngest	39
Average	59.3
Underlying Pulmonary Disease	
Emphysema	21
a. Asthma	20
b. Silicosis	1
Other Heart Disease in Addition to Cor Pulmonale	
Coronary Sclerosis	2
Incidence of Heart Failure	21
X-ray Evidence of Right Ventricular Hypertrophy	14
Electrocardiographic evidence of right ventricular hypertrophy	
Typical	2
Presumptive	8

Correlation:

8 of 14 with x-ray evidence of right ventricular hypertrophy also showed electrocardiographic evidence of right ventricular hypertrophy.

8 of 10 with electrocardiographic evidence of right ventricular hypertrophy showed x-ray evidence of right ventricular hypertrophy.

TABLE II

CLINICAL AND LABORATORY OBSERVATIONS

Hematocrit	
Number of patients	20
Elevated	17
Circulation Time	
Number of Patients	19
Increased	16
Venous Pressure	
Number of patients	19
Increased	12
Vital Capacity	
Number of patients	15
Decreased	15
Cyanosis	18
Clubbing of Fingers	9

TABLE III

FOLLOW UP IN MONTHS

Average time followed from onset of congestive failure to present time 28.1 months

4 followed 48 months or more
8 followed 36 months or more
10 followed 24 months or more
6 followed 12 months or more
6 followed less than 12 months

Four died

1 after 48 months with heart failure
1 after 3 months with heart failure
1 after 2 months with carcinoma of liver
1 after 1 month with empyema

There were 21 patients followed, of whom 18 were males and 3 were females. The average age was 59.3 years. All patients had emphysema as the underlying pulmonary disease, due to asthma in 20 instances, and associated with silicosis in 1. Two patients had coronary sclerosis in addition to cor pulmonale with failure, but in 19 patients no other demonstrable heart disease was present.

All patients had evidence of congestive heart failure.

Fourteen patients presented X-ray evidence and 10 patients presented electrocardiographic evidence of right ventricular hypertrophy. Eight of the 14 patients with X-ray evidence also presented electrocardiographic evidence of right ventricular hypertrophy. On the other hand, only 8 of the patients with electrocardiographic evidence of right ventricle hypertrophy had X-ray evidence of this condition.

Circulation times, venous pressures, and vital capacities were performed as described previously. Circulation times were increased in 16 of 19 patients,

venous pressures were elevated in 12 of 19 patients, and vital capacities were decreased in 15 of 15 patients.

Cyanosis was present in 18, while clubbing of fingers was present in only 9 patients.

The 21 patients were followed from the time of onset of congestive failure until the present. This time averaged 28.1 months. Six patients were followed less than 12 months and 4 were followed 48 months or more. Eight, 10, and 6 patients were followed 36, 24 and 12 months or more, respectively.

Four patients died. They expired 48, 3, 2 months and 1 month after the onset of congestive failure. The first two patients died with congestive failure, the third with carcinoma of the liver, and the fourth with empyema as the cause of death.

This series of 21 patients with cor pulmonale with failure is not large enough to be statistically significant, but at least one observation bears emphasis. The fact that the patients have been followed for an average of 28.1 months following the onset of congestive heart failure, and that several have been followed for over three years, serves to emphasize that these patients are not as hopeless as is generally thought[12]. Instead, with intensive therapy directed towards the pulmonary problem as well as the circulatory problem, patients with cor pulmonale with failure may be expected to live for several years.

SUMMARY

The predisposing factors, and the pathological physiology of cor pulmonale have been discussed. Certain studies have been outlined which are of importance in establishing a diagnosis as well as in following the effects of treatment. The management of cor pulmonale with failure has been outlined, and pertinent data on 21 patients who have been followed for an average of 28.1 months since the onset of congestive failure has been presented, thus emphasizing the fact that the prognosis is not as hopeless as has been previously thought.

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CLINICOPATHOLOGICAL REPORTS

From the Case Records of the Medical College of Virginia
and the University of Virginia Hospitals.

HARRY WALKER, M.D., *Editor*
WILLIAM KAY, M.D., *Associate Editor*

CLINICOPATHOLOGICAL REPORT CASE NO. 86. A 51 year old white male entered the Medical College Hospital because of progessive weakness and weight loss. He had stopped work as a boilermaker approximately six months earlier because of gradually increasing weakness and loss of appetite. About six weeks before admission he began to notice increasing swelling of both legs and shortly thereafter also noted that his urine was dark-colored and that his stools were black. Weakness became so severe that he was forced to go to bed about two weeks before admission. During this time he had lost from 30-40 lbs. He denied having any nausea, vomiting, pain, change in bowel habits, or cough. Chest x-rays three weeks before admission showed some infiltration in the right 2nd interspace and left 4th and 5th interspaces which had improved on a subsequent film taken approximately one week before admission.

The patient had been a fairly heavy drinker prior to the onset of his illness. His mother had died at age 30 with pulmonary tuberculosis. The past and family histories were otherwise non-contributory.

Physical Examination: Temperature 98. Pulse 76. Respiration 20. Blood pressure 114/60. The patient was a gaunt, slightly jaundiced male, appearing much older than stated age. He was somewhat deaf, but mentally clear. The skin showed marked loss of turgor but, except for icterus, was otherwise negative. The teeth were absent, the tongue was dry and smooth, and there were mild excoriations around the mouth. The chest was moderately emphysematous, expanded poorly, and there were atelectatic rales in both lung bases. The heart was normal. The abdomen was scaphoid and non-tender. The liver was palpated three fingers breadth below the right costal margin, was firm and slightly tender. Liver dullness extended upward to the 4th intercostal space anteriorly. The prostate was slightly enlarged and somewhat nodular, but of normal consistency. Feces was dark green in color. There was 2-3+ edema of both legs with thrombotic varices noted in the left calf. The remainder of the physical examination was essentially negative.

Laboratory Data: Urine—amber, acid, 1.020, 1+ albumin, occasional WBC/hpf. RBC 3.5 million, hemoglobin 11.5 grams, WBC 29,200 with 90 polys., 3 eos., 4 lymphs., and 4 monos. The blood smear showed "macrocytic, normochromic RBC with some polychromasia—leukocytosis with increased hypersegmented forms, a few band forms, occasional myelocyte and promyelocyte with eosins increased and toxic granulation present". NPN 44 mgm. %, fasting blood sugar 139 mgms.%. Prothrombin concentration 43%. Serum bilirubin 0.9 direct, total 1.4 mgm. %. Urine urobilinogen was moderately increased and "largely of the levorotatory type". Urine was faintly positive for bile. Cephalin flocculation 3+. Cholesterol 195, esters 94. Total protein 6.6, albumin 2.0, globulin 4.6. Serum acid phosphatase 3.6 B. U. Chest x-ray showed infiltration in the 2nd right and 4th left interspaces. G-I series was reported as showing a constant annular filling defect in the antrum of the stomach 1½ cm. in width and 6-7 cm. from the pylorus. The esophagus and duodenum were normal.

Three days after admission edema of the right leg increased markedly and the right great toe developed a purplish discoloration. At about the same time it was noted that there were purpuric areas over both legs and forearms.

The patient became steadily worse—became confused, incontinent, progessively weaker and oral feedings were impossible. His temperature varied from 97 to 99.8, his pulse from 80-120. The prothrombin concentration remained around 40% in spite of repeated large doses of Vitamin K parenterally. Gastric aspiration revealed free acid and one gastric washing was reported as showing acid fast organisms on direct smear. Subsequent specimens did not reveal acid fast organisms and studies for fungi were negative. Repeated stool specimens were negative for blood and were grossly described as dark brown in color. The NPN rose to 99, the WBC to 40,000 and the patient expired thirteen days after admission. An autopsy was performed.

DISCUSSION BY DR. J. POWELL WILLIAMS*

The first thing that is missing from this protocol, which I always dislike, is an opportunity to know something about the personality and character of the patient with whom we have to deal. That's true not only of our protocol, but of most protocols for CPC's. They deal with the physical rather than the emotional side, a kind of a study of death, whereas in the practice of medicine, this information should form the foundation of any examination. We do know a little bit about this man. We know that his mother died of tuberculosis at the age of 30, and since women in this part of the world don't begin their families until they are 18 or 20, this boy must have been about 10 or 12, if he was the oldest child, younger than this if he were one of the younger ones. In other words, he was raised by a sick mother. In spite of that, if he got tuberculosis at all, it was of the subclinical variety because the past history showed no serious illnesses. There are a few other scraps of information of this type. We know that the man is a boilermaker and that may be of some importance. Boilermakers are proverbially rough, tough he-men who chew tobacco, shoot craps, drink and get drunk, and are hell with the ladies. This is probably the reason that Perdue uses the pseudonym "Boilermakers" for their football team. This man apparently has worked as a boilermaker for quite a while because he is deaf. Deafness is an occupational hazard of living in the turmoil in which these people must exist. That's about all we know about our man. We have to accept him as a pretty solid citizen, a man who feels that sickness is something beneath his dignity, and who would stay on his feet until the last minute. From this we might conclude something about the length of his illness. We know perfectly well that it didn't start six months previously when he quit work. We know that this type of person must have been sick for a good long while, a matter of months probably, and that his total illness was probably a period of a year and may even have exceeded that.

In addition to the duration of illness about which I have already spoken, we know that the onset was very insidious, progression being almost imperceptible with absolutely no evidence of sepsis and no focalizing signs or symptoms. There were only anorexia, progressive weakness, and loss of weight. It is perfectly possible that this man, this character that we have described, ignores such symptoms as gaseous eructations. He probably had had this many times after beer bouts and didn't pay too much attention to it. He may not have been the sensitive individual who makes a fuss about a little "pain in his stomach", but anyway, following the protocol we get a perfect blank all the way through in so far as symptomatology is concerned. We know little or nothing about the interval between quitting work and entering the hospital save that three weeks before admission, he is said to have had dark urine and black stools. If we accept this, it leads directly to an upper G.I. lesion or esophageal varices, but here we begin to get material from the protocol that we really have to cast doubt on. This man, after a year's illness to the point of cachexia, came in with a hemoglobin of 11.5 grams, and had numerous stools examined in the hospital all of which were negative for blood and were recorded as being dark green or dark brown in color. I think that patients are very liable to make mistakes in this observation. If you ask pointedly, "Were your stools black?", and the stools were actually dark green, most patients are likely to answer "yes". In this case, I am inclined to throw it out. We know this though positively, when he finally got to the hospital the complaints that he started with had not changed during a year of progessive illness including the last 13 days of his life—anorexia, weakness and loss of weight, and that's all. Now since we see this man for only the last 13 days of his life, and since you are all fully aware of the fact that during such a terminal period in a cachectic individual, anything can happen—intercurrent infection is the rule rather than the exception and tumors, if present, can appear in places distant from the original site—I think that what we're going to have to do at the start is to review very rapidly the various groups of diseases that might give such nondescript symptomatology as this man presents.

Functional Disorders

I think that we ought to start off with the functional disorders because there are many of them that might present such a picture. The psychotic states, either ordinary depressions or the paranoid type who have delusions about poisoning or the involutional melancholias which are like the depressions, a lack

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of appetite and a refusal to eat are common to them all. These people, as you well know, frequently have to be tube fed for years and years to permit survival. If this man had stayed at home and because of a psychosis simply refused to eat, he would certainly have progressed to a state of asymptomatic starvation. There is another illness which should be mentioned which is more of a personality disorder than a frank psychosis and that is anorexia nervosa in which patients literally attempt to starve themselves to death. We saw one get away with it in our hospital last year. Sometimes they vomit openly and deliberately, but in many instances they eat well and then vomit surreptitiously. I recall also that Dr. St. George Tucker had a boy on his ward who was trying to escape the occupation of steeple jack, which was the one his father had followed all his life and had selected for him. His first effort to escape was to develop falling-out spells. His next effort was suggested by a doctor who told him he had hypoglycemia, which we couldn't confirm, and that he must eat between meals. When we first saw him he was consuming more than 6000 calories a day and losing weight without fever, with a BMR of —20% and entirely asymptomatic. We knew that there had to be a trick in this, but it took several admissions over a period of a year to prove it by assigning attendants to watch him carefully every minute of the day. During this interval he had lost 20 pounds and was on his way to the starvation which the other patient actually died of. A most meticulous autopsy was done on the one that came to death and not one single organic lesion was found. This group of functional disorders we will have to by-pass for lack of any positive evidence in the protocol.

Endocrine Disorders

Of the endocrine disorders that might lead to progressive cachexia, Simmonds' disease or pituitary cachexia is the first that one would think about. That, however, usually occurs in postpartum women and is usually the result of occlusion of the blood supply to the pituitary gland which causes panhypopituitarism. This patient, of course, presents none of the features of polyglandular deficiency. Another disease that may creep up slowly and relatively asymptomatically is diabetes mellitus with progressive loss of weight to the point of emaciation, but usually the patient has polyphagia, polydipsia, and polyuria which this man does not have. He should also have had sugar in the urine and acetonuria in the terminal state, so I think we may discard that. Finally, because he has an increase in his absolute eosinophil count and cachexia, we might think casually of adrenal cortical hypofunction, but we can't consider this seriously because he has nothing else that goes with it. His death was certainly not the hyperpyrexial, paroxysmal affair that we see in Addisonian crisis.

Degenerative Diseases

Considering the degenerative diseases, the conditions that are the greatest killers that we have in these days and times, there is no real evidence that he has any significant degeneration. You do find when you look at his chest film, that the aortic knob is prominent and probably there is some calcification within the wall. We also find that there is a 1+ albumin in his urine, but the specific gravity is 1.020. The heart on the x-ray film is small rather than large, and it is normal to physical examination. There is no evidence of encephalopathy. These are the usual places we look for evidences of degeneration and we find little in this patient. Therefore, we do not believe that the aging process plays any significant part in this man's death.

Collagen Diseases

The collagen diseases I mention because Dr. Porter has so frequently stressed that leukocytosis with eosinophilia should be a red flag to call this group to your attention. The only one of the collagen diseases worth mentioning here, of course, is polyarteritis. This man does have vascular disease, thrombotic disease of the veins of the leg, he has a leukocytosis with a moderate eosinophilia, and he has progessive loss of weight to the point of cachexia. As opposed to entertaining this diagnosis seriously, he has had no fever during any part of his illness, and he has had no neurological involvement which is almost a constant accompaniment of polyarteritis, so I think we will just drop this from consideration and go on. There is one other condition that might be classed with the collagen diseases, namely, Whipple's disease. This lipodystrophy of the gastrointestinal tract is a rare disease but might imitate the condition under study. Without dramatic symptomatology, they usually have diarrhea with fatty stools, but actually a goodly percentage of them do not have diarrhea at all, and may even have constipation. They are unable to absorb fats which are deposited in the intestinal lymph nodes blocking normal drainage.

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Clinically patients go progessively downhill and die in cachexia with little complaint, and for some reason they not infrequently die rather suddenly with no demonstrable precipitating cause. Our patient has none of the other things that go with this diagnosis though. He has no history of arthritic symptoms which may be obtained in the majority of these patients, there is no sign of inflammation of the serous membranes which goes along with this disease, and there are no heart murmurs to suggest a verrucous endocarditis which usually accompanies it, so we will discard it as a serious possibility.

Infectious Diseases

In considering the infectious diseases, we are faced right off the bat with a situation that immediately arouses suspicion. We have a ready-made diagnosis handed to us by Dr. Kay on a silver platter. Here's the history of a childhood exposure to tuberculosis in the susceptible years, because as you all know, the mother prepares all the food and usually supplies most of the affection in the family. It's almost impossible to conceive of this boy growing up without being infected by the mother who had so much tuberculosis that she died of it when the patient was a little boy. We have the insidious onset of his illness which is characteristic of tuberculosis, we have the marked loss of weight, and everything in the clinical picture fits tuberculosis even down to the point of finding the acid-fast organisms in the gastric washings, except for two things it would be most unusual for a patient to go on to death from pulmonary tuberculosis without some of the signs and symptoms of sepsis, such as p.m. temperature, night sweats, chilliness, etc., and there are neither symptoms nor signs of pulmonary disease such as cough or sputum. There is nothing in this whole history to indicate that the patient ever had any fever. As a matter of fact, I called Dr. Kay because I noticed in the last paragraph of the protocol that after the patient had gotten to the point where he had to be fed by vein, when he couldn't be fed by mouth, it is recorded that temperatures ranged from 97 to 99.8, and I wanted to know whether I was right in supposing that these were rectal temperatures. He reviewed the period of hospitalization and reported that there were only three times when his temperature went above 99. The rest of the time his temperature was perfectly normal including the last four days of his life. There was another consideration which forced me to take the diagnosis of tuberculosis seriously. Could this be the disseminated type with involvement of the liver? Twice during the past year we have obtained typical tuberculomatous tissue by needle biopsy of the liver. In one case there was arrested pulmonary disease of longstanding with hepatomegaly and sepsis. In the other there was hepatomegaly and sepsis without evidence of pulmonary disease. The former of these is improving on streptomycin and PAS, but the latter was autopsied and showed diffuse involvement of all organs by tuberculous granulomas. In December in New Orleans I heard the Tulane staff present three cases of sepsis, hepatomegaly, and impaired liver function who showed tuberculous involvement of the liver on needle biopsy but no evidence of pulmonary involvement. Two of their cases had showed marked improvement under treatment with streptomycin and PAS and the third had left the hospital against advice to return later in a terminal state with miliary involvement and meningitis. So if tuberculosis is an acceptable diagnosis, it might also account for his hepatic symptoms and signs, but it must be remembered that in each of the cited cases, sepsis was an integral part of the clinical picture and to reiterate again, in our case it is conspicuous by its absence. Now finally, let's take a look at the chest films which cover a period of about three weeks. In the first film the lesion in the right upper lung field might be tuberculous, but its pyramidal outline with its base toward the periphery looks suspiciously like an infarct and we have in the veins of the lower extremities a perfectly adequate source of embolization. It should also be remembered that infarction may be entirely asymptomatic, without the chest pain, cough, hemoptysis and shock which may be present with large emboli. The lesion at the left base, although less characteristic of tuberculosis or infarction, might be either, but we might reasonably suspect that both are of the same nature. Now when we examine the two later films, we see rather marked resolution in both areas which is compatible with infarction but is not reasonably compatible with pulmonary tuberculosis in the terminal period. In studying these films, I want to call to your attention a very significant finding which is not mentioned in the protocol. If you will notice the lower border of the distal end of the left first rib, you will see that it presents a moth-eaten ap-

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pearance in the first film, further destruction in the second, and finally a large rather clean-cut area of bone destruction 2 to 3 cm. long and 1 to 1½ cm. at its broadest point. This is the osteoclastic type of bone destruction without any evidence of bone production. This is not the picture of osteomyelitis either tuberculous or otherwise, but rather that expected with certain types of malignancy to be discussed later. To sum up, I am going to exclude tuberculosis as the primary illness, in spite of the very excellent evidence presented for the following reasons: (1) The finding of an acid-fast organism by direct smear of gastric washings is such unreliable information that we no longer utilize this examination. The percentage of false positives is so high that we rely only on cultures. There is a bacillus which is commonly found in butter which is acid-fast and even the ubiquitous hay bacillus may lead to such mistakes. No cardia, one of the pathogenic Actinomyces is also acid-fast and fragments of its mycelia might be mistaken for B. tuberculosis. (2) The study of the x-ray films is not suggestive of tuberculosis and does indicate malignancy at some point in the body. (3) Finally, it is inconceivable that our patient has had tuberculosis, either of the disseminated or the pulmonary type, over this long period of time without sepsis, or that he could be dying of this disease while his pulmonary lesions show rapid and definite healing. While it is entirely possible that an active focus of tuberculosis may be found at autopsy, I would think of this as incidental and terminal and not as the primary disease.

THE LIVER

Next I think we should take up the liver not as a focus of intercurrent disease, but as a possible primary entity. It is so obviously a site of disease in this patient. The liver dullness is said to start at the fourth rib and extend three fingerbreadths below the costal margin, which is a good thick liver probably in the neighborhood of 3000 grams. Here again we are presented with a ready-made diagnosis. Our patient is said to be a heavy drinker who gets sick insidiously without sepsis and who finally comes to a terminal cachectic state with a markedly enlarged liver. Superficially this is a pretty good history of cirrhosis, but there are some things that we must take into account here. This heavy drinker I think of as a typical boilermaker, who probably rode through his hangovers without too much difficulty and who was also a heavy eater—he had to be to survive at a tough, hard, physical job. The people who drink a lot and eat a lot rarely get into serious difficulties with the liver. It is much more frequently the ones who start eating whisky instead of food who get into trouble. So while it's possible that this man may have been a heavy drinker, it seems probable that he was also a Gargantuan eater and by this means, protected his liver from serious damage from alcohol. The suggestive history of G. I. hemorrhage—that is the black stools—we have already mentioned and excluded, but there are other negative findings of real importance in deciding whether this liver disease is Laennec's cirrhosis and of assessing its true importance in his total illness. (1) He had no splenic enlargement and he is terminal, within the last 14 days of his life. (2) There are no spider angiomata and no liver palms. (3) There is no evidence of collateral circulation on the abdomen or chest. (4) There is no ascites and the edema of the lower extremities is adequately accounted for both by his thrombotic varices and by his low serum protein. (5) X-ray shows no esophageal varices. In the absence of all these things which go with portal hypertension, I cannot take seriously the possibility that this man is dying of uncomplicated portal cirrhosis. If he has portal cirrhosis, it's not of significant severity and it's not what is killing him, so we still have to go on with our search. There are other kinds of cirrhosis, one Dr. Hennigar hooked me on last year—the so-called postnecrotic cirrhosis. We can say very little about that except that in the history we have here, there is no evidence anywhere of an acute necrotic episode which this so-called postnecrotic cirrhosis should follow. The slow chronic hepatitis which may be subclinical and may follow a subclinical viral infection, ends up with a perfectly typical picture of Laennec's cirrhosis just as most of the postnecrotic type do, so that I think we can rule out and cast aside both of these simultaneously. Let's approach it from another angle now. What's the commonest cause of death in cirrhosis? It is intercurrent infection with focal necrosis of the remaining normal liver tissue and cholemia. That accounts for about 35 per cent of the deaths. About 25 per cent of them die of hemorrhage and around 20 to 25 per cent of them die of primary cholemia without nec-

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essarily having the focal necrosis. The cirrhotic process simply destroys more liver tissue than coincidental reparative processes can replace and death ensues. The death described here doesn't conform to any of these mechanisms.

Now let's attack it from the standpoint of the degree of functional disability. Does the hepatogram give evidence of terminal liver disease? The most significant thing is the serum protein. There is an albumin fraction of only two grams which is a very decided reduction, and along with it there is elevation of the globulin. That is very characteristic of severe liver disease. Since shifts in the proteins don't take place rapidly, it would tend to indicate a rather prolonged impairment of liver function as well as a reasonable degree of severity unless we take into account one other thing. This man has been starving for a year to the point of cachexia. We know that the serum protein in the true starvation state may be low from an inadequate intake of materials with which to build albumin, and further, that starvation per se will lower both the liver's efficiency and its ability to maintain its homeostasis, hence starvation may be a large factor in producing this clinical picture. The cholesterol was of reasonably normal value though the esters are somewhat below the normal. The cephalin flocculation was reported as 3+. I think we should remember in evaluating the cephalin flocculation that it is not a specific test. We found overseas when we were doing them by the hundreds and thousands, that the cephalin flocculation was positive in many infectious conditions and the incidence was particularly high in cases of atypical pneumonia. In addition, we also found that the cephalin flocculation was a very very sensitive test. To give you an example, we found that people with chronic hepatomegaly almost invariably had a normal cephalin flocculation before peritoneoscopy and biopsy and a 4+ flocculation the day following. So it takes a very small amount of liver damage to make the test become positive and it will be positive for things other than liver disease. At any rate, the 3+ is not what one might expect in a terminal liver case. The prothrombin time was 43%. This certainly is a moderate reduction. You all know that in treating your occlusive cardiovascular diseases, you usually force the prothrombin concentration down to about 25 per cent and hold it there. The thing that is more significant about the prothrombin though is the fact that although vitamin K was given in perfectly adequate doses, this man's prothrombin time did not go up, which is pretty definite evidence of liver damage. The serum bilirubin is rather interesting. The elevation is very slight. I don't know what you take as normal down here, but we usually accepted as normal a reading as high as 1 mg. per 100 cc. In this patient it is 1.4 mg. In other words, there is a slight elevation. Of this total 0.9 mg. is the direct reacting or one minute bilirubin which means that it has passed through functioning liver cells, which were able to convert it to the direct reacting sodium bilirubinate. Also, we note that the urobilinogen is reported as moderately increased. The source of urobilinogen is bilirubin so that at least there was enough functioning liver tissue to put more than the average normal amount of bilirubin from the blood stream into the bowel. The macrocytic normochromic anemia fits in perfectly well, the slightly elevated blood sugar of 139 mg.% fasting might be thought to be due to liver damage, but to sum up, this clinical picture is certainly not that of terminal liver disease. It is rather the picture of mild intrahepatic obstruction probably from some infiltrative disease or fibrosis and we're still going to have to go ahead and try to find out what it is that's keeping this liver from doing its work normally. As you all know, it takes only about 20 per cent of functioning tissue to do the work of the liver, but again, this is not the picture of terminal liver disease. This man is not dying of liver damage.

Malignancy

This brings us to the group of diseases I have saved for last. What group of diseases are by far the most likely to cause the clinical picture here presented from start to termination? Beginning insidiously in a robust man of 50 with almost imperceptible progression over a period of a year or more and characterized only by anorexia, weakness and weight loss with a complete absence of septic symptoms and no focalizing signs until the terminal state? The answer is only too obvious, malignancy. Here again we are offered a wide choice of sites for the primary lesion. We have the prostate, the stomach and the lung, and still others on the x-ray films but not mentioned in the protocol. Let's look at the x-ray films of the gastrointestinal tract.

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DR. MANDEVILLE*: As far as I am concerned, I can see some mucosal relief where there is a somewhat pinched-off area in the stomach, but I would not favor this as carcinoma or as being intrinsic in the stomach. This man was of heavy build and heavily built people do have their stomachs more anteriorly than slim people, although he is supposed to have had a scaphoid abdomen. He had lost some forty pounds of weight, however, to account for this scaphoid abdomen, but there can very well be some anterior displacement of this stomach by a pancreas or by nodes, there could be an aberrant pancreas causing this annular defect, there could be pancreatic tissue in the stomach wall which would not ruin the mucosal relief. Retroperitoneal malignancy certainly cannot be excluded by these x-ray films. I'm not in favor of an intrinsic carcinoma, however. Dr. Williams has some films here of what he considers to be a normal stomach showing that there is no anterior displacement on the right and left lateral views of this stomach which he wants shown for comparison with the lateral views of the stomach seen in this case.

DR. WILLIAMS: I'd like to correct Dr. Mandeville. This isn't my opinion, it is my x-ray adviser's opinion. I went to him and asked him for a normal lateral view of the stomach and this is what I got to compare with the films on this patient. Now, as Dr. Mandeville has said, his man has lost 40 pounds of weight or more and is described as having a scaphoid nontender abdomen. How in the world then can his stomach defy gravity and sit out that far away from the vertebral column unless there is something behind it pushing it out. This "annular constriction" looks more to me like the pylorus and I believe it is the type of annular constriction that you and I have and I hope we'll keep. So we'll just discard the stomach from any consideration right now in spite of the protocol. I'm going to accept the premise that this man has a mass, a retroperitoneal mass, which had displaced his stomach anteriorly, which is not recorded in the protocol, and I want you to remember the possible metastatic lesion in the first rib, which is also not in the protocol.

Malignant Disease

In discussing malignant disease, I think we'll have to do a thorough job just as we tried to do in the other conditions considered but will mention only briefly a few things at the start such as leukemia because of the very high count though if you notice, the count from a qualitative standpoint shows that the shift is decidedly to the right with many hypersegmented forms. This is just the opposite of leukemia. Though leukemia may cause these lesions of the lungs and liver, we will discard it for lack of evidence. The one thing that we do get from the leukocyte count is the type of picture which may be compatible either with malignancy or with infection. The lymphoblastomatous diseases were considered for a while as a possible explanation for the lesion in the stomach until I convinced myself that this was almost certainly a broad pyloric ring with perfectly normal mucosal pattern. I was thinking of an intramural lesion which did not involve the mucous membrane because you can see the mucous membrane perfectly smoothly over that gap. As a matter of fact, it is so smooth that it might even suggest an atrophic gastritis more or less similar to the atrophic tongue that he has, which, along with the excoriation of his mouth, is, of course, an indication of avitaminosis, a part of his starvation. Hodgkin's disease can give a blood count of 29,000 with eosinophilia. However, he has no palpable peripheral nodes and the spleen is not enlarged. He does have a retroperitoneal mass. Therefore, he might be looked upon as compatible with the Pel-Ebstein type of retroperitoneal Hodgkin's disease which does occur with no evidence of lymphoid hyperplasia elsewhere. But he has no fever—we come back to that over and over again—and fever is the most distinguishing characteristic of the Pel-Ebstein type. It may be intermittent at first, but when they come to die it usually becomes continuous. Incidentally, Hodgkin's disease will attack bones too, chiefly in an osteoclastic way just as is the case with the first rib I called to your attention, but because of the absence of fever, and the several other deficiencies noted, we will discard Hodgkin's disease.

Prostate

Of the carcinomas, let's start first with the least likely ones. We've already thrown the stomach out of serious consideration, in spite of the bait offered by the protocol. Let's now go back to the prostate because it was said to be of normal consistency but nodular and the acid phosphatase was 3.6 Bodansky units, which is fractionally above the book level of normal. Most G. U. people don't accept 3 units as

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normal. They don't get concerned about the acid phosphatase until values exceed 5 Bodansky units and I'm not enough of an authority to have an opinion of my own. I do know, however, that when carcinoma of the prostate metastasizes to bones, it usually goes to those of the pelvis and lower back. I know also that these metastases are almost invariably of the mixed type, that is, bone destruction along with bone regeneration which is not the kind of metastasis that we see in the first rib. Our G. U. colleagues, aided and abetted by our pathologists, tell us that about 60 per cent of men in this age group have carcinoma of the prostate. We clinicians know that this is not true in the strictest sense, and suspect a misuse of the term "carcinoma". By their criteria, our patient may have their kind of "carcinoma", but we as clinicians are certain that whether he does or not is of little consequence in his total illness and so, we'll drop the prostate from further consideration.

Primary Carcinoma of the Liver

Now for the liver. In primary carcinoma of the liver, metastases are the exception rather than the rule and when they do occur, involve chiefly the regional lymph nodes and the lungs. Distant metastases, such as the first rib in this case, are very rare. Those which reach the lungs usually do so by eroding directly into a portal radicle forming carcinomatous thrombi with later embolism. This could be a primary carcinoma of the liver, but I doubt it. I doubt it because in the first place, this is such a rare condition, and secondly, because I think we have a much better explanation.

The Lung and the Pancreas

The other two primary foci that deserve serious consideration are the lung and the pancreas. I doubt that the lesions we see in the chest x-ray are those of carcinoma because they show such definite evidence of regression over a period of three weeks, which would not be expected of carcinoma, either primary or secondary. However, we recently autopsied a man 59 years of age who came into the hospital with the complaint of back pain of three weeks' duration. A routine chest film showed an area of atelectasis at the base of the right lung along the right border of the heart. The patient grew worse so rapidly that we never got to bronchoscopic examination. Incidentally, he had marked leukocytosis and eosinophilia, varying from 19 per cent to 43 per cent, much higher than in this case. When autopsied, his liver was found to be studded with metastatic nodules and the retroperitoneal nodes were extensively involved. The primary site was in the posterior branch of the right lower lobe bronchus, was about the size of the end of your finger, and could not be seen on the chest x-ray. So it is possible that this did come from the lung. In deciding between carcinoma of the lung and carcinoma of the pancreas, I thought that I had a differential point in that thrombosis of veins over the body and psychotic states were commonly found with pancreatic lesions. However, on November 24 the American Medical Journal contained a very good article on the subject of venous thromboses in malignancy, which I commend to your attention. The author reviews the literature to show that multiple thromboses may occur with malignancy in any organ and cites four of his own cases of carcinoma of the lung in which the initial complaint was of multiple thromboses, so my differential point was gone. I thought I had another differential point in that the only cases of metastases to the bones from pancreatic tumors that I could recall were of the osteoblastic variety, that is, with a lot of bone regeneration along with destruction. I finally found a book written in 1951 on tumors of the pancreas by Dr. Maxwell H. Popple of Mt. Sinai Hospital in New York, which stated that 6.3 per cent of pancreatic carcinomas metastasize to bone and that the metastases could be either osteoblastic or osteoclastic. So again my differential point disappears and I find myself thrown right back on the law of probability and chance. We do know that the most frequent cause of the clinical picture we see here is by long odds carcinoma of the pancreas, when lesions of the rest of the gastrointestinal tract have been excluded. A small bronchogenic carcinoma could give the same picture as in the case cited, but on the basis of statistics alone, the pancreas is much the best bet. The clinical picture is typical of involvement of the body and tail, and in all cases of carcinoma involving the liver, where the stomach is ruled out as a source, as well as the large bowel, the pancreas is the best bet nine times out of ten.

Therefore, I am going to give as my first choice carcinoma of the pancreas involving chiefly the body and tail, since there is little or no evidence of common duct obstruction, with metastasis to the left first rib, to the retroperitoneal nodes, and with multiple

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metastases to the liver. There may be also some so-called carcinomatous cirrhosis in the liver, that is, the reaction to a foreign body. We know also that he has hypertrophic arthritis and a moderate degree of arteriosclerosis. He may also have moderate nephrosclerosis to account for his 1+ albumin. The lung lesions are probably infarcts though a terminal tuberculosis would not surprise me.

DR. JAMES BURKE*: I liked Dr. Williams' discussion very much. My impression was that the patient had carcinoma of the pancreas and I thought at the time that it was unusual that he had no pain. Pain, as you know, is present in about 90% of cases of carcinoma of the pancreas. There was no history of pain from this man though it is possible that it was because of his terminal condition that we were unable to elicit the history of pain. I am not sure.

HOSPITAL DIAGNOSIS: Carcinoma of Pancreas.

DR. WILLIAMS' DIAGNOSIS: Carcinoma of body and tail of pancreas with metastasis to the left first rib, retroperitoneal nodes, and liver.

PATHOLOGICAL REPORT: DR. W. GLEN REED†

The body for autopsy was that of a poorly nourished white male, appearing a little older than sixty years. There was pitting edema of both legs to the thighs. The right great toe was dry and gangrenous.

The heart was not remarkable except for some coronary sclerosis. The aorta was the seat of marked atherosclerosis. Two abscesses, each five centimeters in diameter, were found in the right middle lobe and in the left lower lobe of the lung, respectively.

The abdominal cavity contained a few thin, fibrinous adhesions and about two hundred cubic centimeters of a clear, yellow fluid. Plaque-like tumor nodules were seen on the peritoneal surface of the diaphragm. The liver weighed two thousand, seven hundred grams and was studded by sharply delimited nodules of firm white tissue, ranging up to four centimeters in diameter. These frequently had necrotic or hemorrhagic centers. The biliary tract was patent, but a tumor plaque was present on the serosal surface of the gall bladder. At the upper pole of the spleen, a firm, pale, well defined infarct was found. The left kidney also showed a small infarction.

A shallow, hemorrhagic duodenal ulceration, seven tenths of a centimeter in diameter was found over the head of the pancreas. The body and a portion of the head of the pancreas were replaced by a firm, cream-colored mass of tissue, mottled with hemorrhage. The distal portion of the pancreas was scarred and atrophic.

Thrombi were found in many pancreatic vessels, in the splenic vein, portal vein, iliac veins, and femoral veins.

The prostate was small, firm, yellow-tan, and nodular.

Microscopically, the tissue of the head and body of the pancreas had been replaced by pleomorphic tumor cells. (Fig. 1) These cells were arranged

Fig. 1.—View of the carcinoma in the pancreas, showing the arrangement of strands and imperfect glandular structures well supported by stroma.

in small clumps and strands, or were seen lying singly. Occasional tiny, imperfect glandular formations were noted. The tumor was very well supported by fibrous stroma which widely separated many cellular groups. Some of the cells attained giant proportions.

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The carcinoma had invaded the mucosa of the duodenum at the point of gross ulceration. There was a generalized thrombosis of the arteries and veins of the pancreas. Tumor plugs were noted in many peri-neural lymphatics. (Fig. 2)

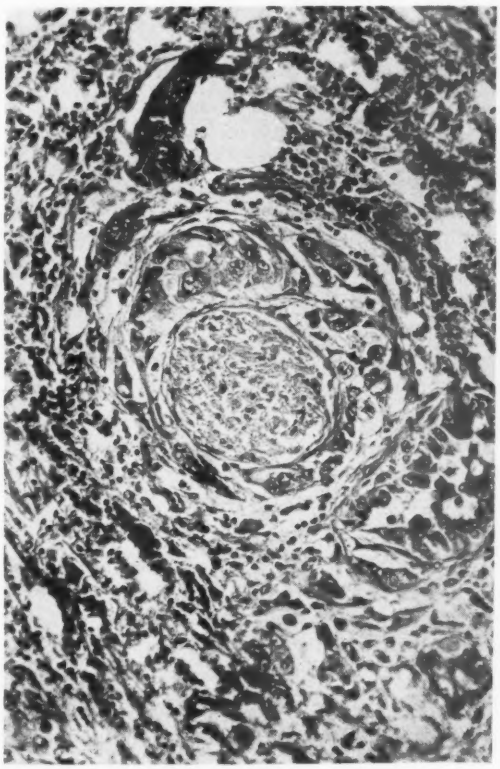

Fig. 2.—A peri-neural lymphatic, in the pancreas, filled with tumor cells.

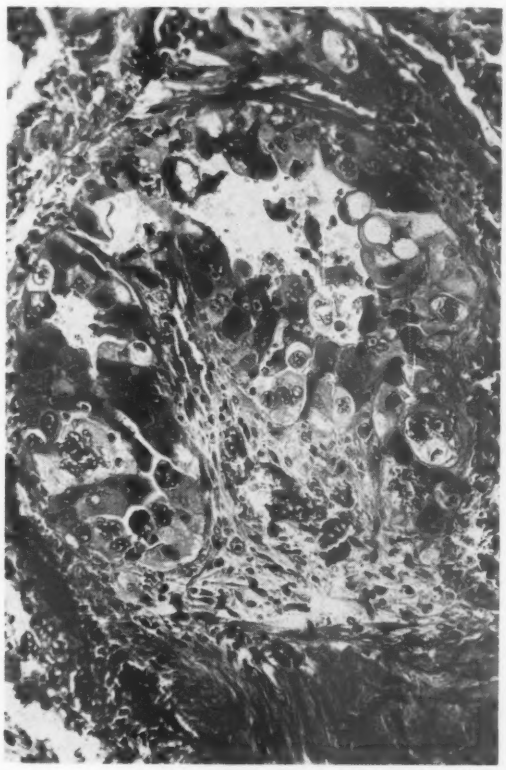

Fig. 3.—Tumor and partially organized thrombus, filling a blood vessel in the lung.

Wide spread metastases were found, some in organs which showed nothing grossly. Numerous small carcinomatous foci were present in the lungs, lying in alveoli, blood vessels, (Fig. 3) and lymphatics. Near one of the abscesses, organized arterial thrombi were encountered, making it highly probable that the lesion originally was an infarct.

The majority of the metastatic nodules in the liver had undergone central necrosis. The spleen showed a typical necrotic infarcted area. The left kidney, in addition to its infarct, revealed a glomerulus containing pleomyorphic cells from the pancreas. Several tumor giant cells were discovered in the tiny vessels of the brain.

The thrombi in the larger veins (iliac, femoral, splenic) were dry, firm, and laminated. An incidental finding, in this case, was an adenocarcinoma of the prostate.

Several features are of interest in carcinoma of the body and tail of the pancreas. Its correct and early diagnosis is difficult and rarely made, due to the vague and non-localizing signs and symptoms. It does not betray itself, early, by jaundice, as does carcinoma of the head of the pancreas. The most constant presenting symptom is pain in the abdomen, less frequently involving the lumbar region.

Carcinoma of the body and tail of the pancreas metastasizes more rapidly and widely than does carcinoma of the head. The head of the pancreas is bounded by the duodenum, transverse colon, and posterior abdominal wall, all relatively resistant to tumor invasion. On the other hand, the anterior and inferior surfaces of the body and tail are covered only by peritoneum. Thus, in its unobstructed local spread, it reaches many blood vessels and lymphatic channels

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and disseminates widely. Furthermore, the body and tail are drained by the lienal vein, which is less easily blocked by tumor than are the numerous small veins of the head.

The association of multiple venous thrombi with carcinoma of the tail and body has been reported, but not satisfactorily explained. In the absence of other localizing signs, multiple venous thromboses may be an early clue to this diagnosis.

Finally, mental changes have been noted with the tumor, and a direct correlation has been postulated. However, the patients present such a diagnostic problem, and are so frequently labeled as psycho-neurotics, that they perhaps are entitled to a few mental aberrations.

Pathological diagnosis. 1. Adenocarcinoma of body of pancreas with generalized metastasis. 2. Multiple venous thrombosis. 3. Adenocarcinoma of prostate, incidental.

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Conferences for Chaplains for Mental Hospitals.

Several Veterans Administration hospitals are conducting conferences and clinics for the clergy to increase their understanding of the mentally ill. This program is part of the VA nation-wide policy to acquaint the public generally with the fact that the mentally ill can be rehabilitated as self-supporting citizens with good medical care and the understanding of their home communities.

The interest developed in building closer working relationships between members of the medical profession and the clergy has led to many of these VA conferences being put on an annual basis. In some cases, VA hospitals have had to conduct more than one conference in order to accommodate all of the clergy in the neighboring area who wanted to attend. Chaplains at the VA hospitals and members of the psychiatric staffs conduct the conferences and clinics. Although the conferences and clinics are the responsibility of each hospital manager, they are encouraged by the VA Central Office in Washington, D. C.

Not all conferences and clinics are confined to the subject of the mentally ill. Some are conducted to improve hospital-community relations so as to increase understanding of mutual responsibilities in the care of the ill. The meetings are attended by the clergy of all faiths. In one case, they came from communities as far as 80 miles from the VA hospital conducting the conference.

The scope of the problem in increasing the general understanding of the mentally ill is reflected in the latest VA statistics. Of the 101,519 VA patients on March 18, 1952, more than half, or 51,940, were classified as neuropyschiatric patients. And of the 51,940 NP patients, 45,509, or nearly 90 per cent of the total, were classified as psychotics. The remaining 6,431 patients were classified as other psychiatric and neurological patients.

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THE MEDICAL SOCIETY OF VIRGINIA

PUBLIC RELATIONS

Report of the Annual Public Relations Conference

The Annual Public Relations Conference of The Medical Society of Virginia was held in Richmond on April 10, 1952, at the John Marshall Hotel.

Woven around the theme, "Let's Step Up Our Public Relations", the program featured outstanding morning speakers, a luncheon address by Virginia's Lieutenant Governor, and two interesting panel discussions in the afternoon.

Dr. John T. T. Hundley, President of the Society, served as Conference Chairman and opened the meeting with a word of welcome to the group of seventy.

The Public Relations Program and Objectives of 1952 were outlined by Dr. James P. King, Radford, Chairman of the Public Relations Committee of The Medical Society of Virginia. He scored the seeming lack of "Public Relations Consciousness" among many members of the Society, and emphasized the need of a Public Relations Committee in every component society.

Dr. King stressed the part the Public Relations Committee has played in the promotion of the A.M.A. plaque, "To All My Patients". He commented that, "Doctors certainly need to tell the patient more about their fees, and especially the big or unusual ones."

The next speaker was Leo Brown, Director of Public Relations of the A.M.A., who told the group that the "unethical or thoughtless act" of just one physician leaves its scars on the entire profession.

He called for night emergency call systems in every community, one of the best ways to answer the often heard complaint that a doctor was not available.

Mr. Brown believed that component societies should hold special meetings designed to revive the interest of old members and to fully acquaint new members with the problems of the day.

In concluding, he stated that we right now have the best medical service system in the world, but it must not be taken for granted. Rather, we must constantly seek to strengthen it.

Miss Charlotte Rickman, Raleigh, North Carolina, Consultant on Rural Health for The Medical Society of North Carolina, gave a brief history of rural health activities in North Carolina. At the present time there are thirty-eight rural health councils in the state, and they are all active.

Miss Rickman reported that no one county can set a pattern for others. Needs and problems vary, and people think differently. However, they all cite better

Conference Luncheon

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(L to R) Dr. J. P. King, Public Relations Chairman; Mr. Leo E. Brown, AMA's Director of Public Relations; Dr. J. T. T. Hundley, Conference Chairman; Miss Charlotte Rickman, Rural Health Consultant, North Carolina; Dr. Harlan English, Rural Medical Service, Illinois, head morning session.

sanitation and a community house as standard needs.

It was brought out that in most instances, it takes time to get rural people together and some action started. However, once they become interested, they are hard workers and have a knack of getting things accomplished.

Climaxing the morning program was a talk by Dr. Harlan English, Danville, Illinois, dealing with "Money and Doctors' Fees". He pointed out that the cost of medical care has risen 48 per cent in the last fifteen years, while the overall cost of living has increased 72 per cent.

Dr. English recommended that physicians pay close attention to their accounts. It was suggested that a definite understanding be had with the patient about fees, billing times and procedures, etc. In this way, risk is almost completely removed if physicians will stick to the arrangements agreed upon.

It was the opinion of Dr. English that no physician should charge a person more than one month's income after taxes for any type of operation.

Luncheon was featured by an address by Lieutenant Governor Lewis Preston Collins, in which a plea was made for more emphasis on "life's intangible values—morals, ethics, and a sense of legal responsibility."

He urged physicians "to get back to the basic influences exercised by the old country doctor", and declared that "by reason of his influence the country doctor was able to bring about a greater individual and social consciousness of the reality and the necessity of intangible values."

The first panel discussion of the afternoon session was on the subject, "Can We Improve Our Voluntary Health Plans?" The moderator was Dr. John O. Boyd, Jr., Roanoke, and the panel members were Dr. William R. Pretlow, Dr. Benjamin W. Rawles, Jr., and Dr. Ennion S. Williams.

Dr. Boyd reported that an attempt is being made by the Medical Service Committee of The Medical Society of Virginia to formulate a program by which the coverage now available could be improved. There is, he said, a great public demand for increased coverage and medical care.

It was the opinion of Dr. Rawles that while the medical profession should promote Blue Cross-Blue

(L to R) Dr. J. L. Hamner, Mr. Leo E. Brown, and Dr. J. T. T. Hundley chat with luncheon speaker Lieutenant Governor Lewis Preston Collins (second from right).

Shield, it should also give a helping hand to the policies of private indemnity companies. However, physicians should be sure patients are getting that for which they pay.

Several methods were suggested by Dr. Rawles for the promotion of voluntary health plans. The best promotion job can be done in the doctor's office with the physician explaining the policies to the patient. Of course, physicians should understand voluntary health insurance thoroughly in order that a proper selling job may be accomplished.

Dr. Pretlow pointed out that Blue Cross is still a very young organization, and actually still has much to learn.

He quoted statistics to show that many persons are using Blue Cross for purposes not essential, and unless this practice is ceased, Blue Cross can be priced out of the market. It was emphasized that physicians must strive to hold down nonessential hospital admissions.

The necessity of opposing socialistic trends in all fields of endeavor was stressed by Dr. Williams. He stated that compulsory health insurance is but one objective in the campaign to socialize America.

The concluding panel dealt with the "Public Relations For the Component Society", and was moderated by Dr. Marcellus Johnson, Jr., Roanoke. Panel members were Dr. Fletcher J. Wright, Jr., Dr. Edward E. Haddock, and Dr. W. Callier Salley.

Dr. Johnson described the efforts of the Roanoke Academy of Medicine in getting its medical public relations program under way. He cited the need of having some younger men on public relations committees, in order that necessary training might be had. It was pointed out that these younger members will be of tremendous value to the medical profession as time goes on.

Deploring the fact that the medical profession has allowed an insurmountable wall to be built about it, Dr. Johnson explained that the profession is now being awakened by the blows which have been dealt from all sides. It is now more important than ever that each physician become a public relations committee of one.

Dr. Wright told how important it is that physicians take an active part in civic life. Registering and voting are "musts", and accepting civic responsibility is the duty of every doctor.

According to Dr. Salley, the medical profession must work closely with other reputable organizations and groups whose aims are similar to our own. Most of these organizations have indicated a desire to cooperate with the medical profession.

The need of a good Doctor-Press-Radio relation-

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PR exhibit displayed at conference.

ship was pointed out by Dr. Haddock. Other states have achieved noteworthy results, and there is no reason why our component societies can't do the same.

In this connection, Dr. Haddock reported the efforts of the Public Relations Committee of the Richmond Academy of Medicine. Talks held with representatives of the press and radio brought forth many interesting and helpful comments, and a better understanding now exists as a result.

ROBERT I. HOWARD, *Director, Public Relations, The Medical Society of Virginia.*

TEN WAYS WE CAN WORK INDIVIDUALLY FOR BETTER PUBLIC AND PROFESSIONAL RELATIONS

1. Be civic-minded and active in community affairs.
2. Schedule appointments we can meet on time.
3. Itemize our bills.
4. Explain to patient in understandable terms why an expensive drug is prescribed and what results may be expected.
5. Urge hospitals to minimize routine costs.
6. Invite patients to discuss our fees; display the AMA plaque "To All My Patients."
7. Encourage Voluntary Health Insurance Plans.
8. Pay special attention to reception room. Keep it neat and comfortable.
9. Supply sound PR literature to patients.
10. Urge office personnel to be PR minded.

OUR PUBLIC RELATIONS PROGRAM AND OBJECTIVES FOR 1952

JAMES P. KING, M.D.,
Chairman, Public Relations Committee,
The Medical Society of Virginia
Radford, Virginia

The wheels of our public relations program all but came to a standstill in 1951, following the untimely death of our beloved director, Henry S. Johnson. To get the program underway once more, it became necessary to appoint a successor to him, who assumed office at our annual meeting last fall; also to name several new members of the Public Relations Committee. Since then, a re-formulation of the program has been and still is underway. Several projects have been advanced and completed during this period and many others are under advisement. In carrying forward your public relations work, we have made extensive use of that well-prepared document, OBJECTIVES AND PROJECTS OF THE DEPARTMENT OF PUBLIC RELATIONS OF THE AMA, the author of which, Mr. Leo F. Brown, is one of our guests here today. His publication has guided us in the selection of things that we believed could be accomplished in our State. Many of you have copies of this outline and I urge you to study them thoroughly. In my opinion, if we were able to implement in its entirety a program such as Mr. Brown suggests, all over this country, we should probably find it unnecessary to meet here today.

In order to further our own education regarding this aspect of our profession and to broaden our perspective, Secretary Bob Howard and I traveled to Los Angeles last December to attend the AMA's annual Public Relations Conference, a well-planned and well-executed meeting, from which we received many valuable ideas for our work in Virginia. At that time we became acutely aware of the need to inform and interest more doctors in public relations work. As this awareness has grown we have been amazed to witness the lack of what may be called "P-R consciousness" among many members of our medical society. A number of these physicians are not only indifferent to, but are actually against, public relations programs because they believe them to be unnecessary. So it seems that we need first to stir up and maintain interest among our own colleagues. To do this, we believe that a public relations committee should be formed in every component society in the State. Thus, during the past several months, the officers of each of these societies have been requested to see that such a committee is organized or reactivated. It follows that, once inaugurated, each committee should take an aggressive role in its society, presenting at least one public relations program every year, seeing that special societies are actively engaged in some public relations work, and keeping its members informed and properly oriented in all matters pertaining to the medical profession and its contacts with the public. Also, to help inform and interest the individual doctor, CURRENT CURRENTS is being sent out every six weeks. I believe that you will agree that this publication has shown much improvement in recent issues in the quality and appeal of the articles it contains.

It has been aptly said that good public relations begin and end in the doctor's office. In keeping with this view, your Public Relations Committee believes that practically all effective work in this field should be on a local level. Accordingly, our effort is being exerted most strongly in that direction. At all times, we wish to stress good doctor-patient relationship, as the following report of our activities will attest.

Here in Virginia we are getting our message over to the public through considerable use of the radio. We have recently completed a series of seven recorded broadcasts over Richmond's 50,000-watt station, WRVA. The series entitled, "Meet Your Doctor", was designed to acquaint the public with medical problems and services in Virginia. Dr. Ennion Williams of Richmond has served as chairman of this committee. Eight well-qualified physicians, from various sections of the State, were chosen to participate in the programs. From all available information, the broadcasts have been very well received. One evidence that such has been the case has been the receipt of inquires about them from the headquarters of the AMA and from one of the larger drug houses in the country. This series is available

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for re-broadcast in other areas of the State; you should arrange with Secretary Howard for your local station to carry the programs.

Because the role of the medical secretary, office receptionist, or nurse in the doctor's office has received increasing recognition during the past year, your Committee has approved special bulletins intended for members of this group, who have so very much to do with the doctor's impression upon his public. A series of lectures on this aspect of public relations has been prepared and can be given to groups of medical assistants by personnel of our Richmond office.

Another important accomplishment of your committee has been the assembling and distribution of a kit of materials to all component societies describing services used throughout the country for taking care of night and emergency calls. As you know, instances have happened in at least two cities in Virginia that have brought down upon our heads the wrath of the press. Regardless of the merits of these incidents, the public's reaction has been somewhat unfavorable to the medical profession. We believe that this is a good place for preventive work; that all medical groups throughout the State should arrange for adequate handling of emergencies and should not permit isolated instances of mismanagement to overshadow the commendable service that physicians everywhere render emergency patients.

A concentrated effort has been made to promote the use of the AMA plaque, "To All My Patients". We have received reports from physicians who have used the plaque and they tell us that the simple invitation to discuss fees has often resulted in opening the conversation without embarrassment to either the patient or the doctor. Incidentally, I am told that we are well ahead of our neighboring states in promoting the sale of this plaque.

Your Public Relations Committee is currently interested in the betterment of doctor-press-radio relations. We believe that many of the unfavorable shadows cast upon the medical profession by the press and radio can be removed by a mutually helpful code of cooperation. This has been true in many states, and in a few cities in Virginia, where local committees have sought working arrangements with the press and radio. Dr. Haddock will have more to say about this subject later in the program. Perhaps many of you have "Calendar Suggestions for P-R Committees" prepared by Bob Howard and colleagues and recently sent to all presidents and secretaries of component societies. This booklet explains, in greater detail than I can undertake, many of our projects and objectives.

More public relations material and information has been sent to physicians and member societies during the past six months than in any comparable period in the history of The Medical Society of Virginia. A real effort is being made to keep you members informed. We hope that ours will not be a one-way information track, but that we may have your candid views at frequent intervals.

There are almost unlimited fields of endeavor for local committees and component societies who want to work for improvement of their relations with the public. The ten ways we can work individually, ways which are listed in your program today, are enough to keep us all busy for a long time. As we look over this list we realize that we need more physicians in public life: as members of city councils, as leaders of community drives, and as participants in various other worthwhile civic enterprises. We should remember that time is money to the patient, too. One of the speakers in Los Angeles talked on this subject and he began by observing complete silence for a full minute. The audience noticeably squirmed and when he did begin he said "It's irritating, isn't it? "Doctors certainly need to tell the patient more about their fees and especially the big or unusual ones. For many years, it has been the custom at the hospital of my affiliation that, before the patient is admitted, we explain the charges—all of them—and actually give his family an itemized sheet showing these expected charges. As a result, very few misunderstandings arise regarding fees and other charges. I believe this system could be followed with advantage by every physician, especially when the fees may be substantial, unusual or unexpected.

I have read and re-read the *Objectives and Projects of the AMA* to which I referred a few minutes ago and as I have done so each item has seemed the most important one. Chairmen of local public relations committees will profit by careful study and utilization of this master plan.

During the past few years the medical profession has frequently found itself in deep water, fighting socialized medicine with a back-to-the-wall spirit.

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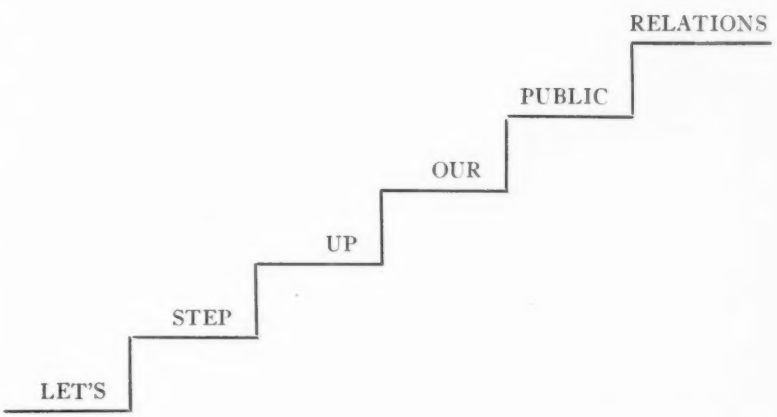
After some successes, such as occurred in the senatorial races of 1950, it appeared that we might once again settle down to the serious business of practicing medicine without the threat of governmental interference. Unfortunately, our leaders in government (Messrs. Truman, Ewing et al) have not seemed content to let us do so. We have repeatedly observed the attempt to enact into the law the so-called fringe bills; free hospial care of the aged, Federal Aid to Medical Education, and, most recently, the Emergency Maternal and Infant Care bills, all of which are parts of the scheme of the national planners to invade the field of the practice of medicine. Further, we have heard and read the public expressions of members of the present federal administration concerning compulsory medical taxation. Thus, we realize more keenly than ever that our profession, which has unquestionably made American medicine the finest in the world, must find means of providing its benefits to *all* our people, irrespective of economic status; that until *we* do so, we are going to have the constant threat of governmental interference. We must remove the cause if we are to prevent the malady. As our new Health Commissioner said recently, "We must put a shingle on the roof rather than a bucket under the drip." Until this ideal of serving all our people can be accomplished, we need to use the keenest tools at our command to counteract the malignancy of socialized medicine. Among such tools, some of the most important are those to be found in good public relations programs throughout the Nation.



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GETTING CLOSER

SEPTEMBER 28—OCTOBER 1

Those are the dates on which the Annual Meeting of The Medical Society of Virginia will be held. The place, of course, is Richmond, and headquarters will be the Hotel Jefferson.

If you haven't made your reservations, do so now, by writing any of the hotels listed below.

JEFFERSON* Headquarters Hotel	FRANKLIN & JEFFERSON
JOHN MARSHALL	5TH & FRANKLIN
KING CARTER	8TH & BROAD
RALEIGH	9TH & BANK
RICHMOND	9TH & GRACE
WILLIAM BYRD	2501 WEST BROAD

* The House of Delegates of The Medical Society of Virginia has directed that officers and delegates of the Society shall be given the opportunity of securing reservations at the Headquarters Hotel. However, the Jefferson has guaranteed the Society a total of 250 rooms, and reservations for the general membership will be confirmed in the order in which requests are received.

MISCELLANEOUS

V.A.G.P. Scientific Assembly Highly Successful.

Meeting in Roanoke for their Second Annual Scientific Assembly, May 8-9, the Virginia Academy of General Practice chalked up another record for an eminently successful Scientific Session, largely attended by both General Practitioners and Specialists in other fields of medicine.

Comprising outstanding men in their several fields, the speakers, gathered from the teaching and hospital staffs of the two Virginia Medical Schools—the Medical College of Virginia in Richmond and the University of Virginia in Charlottesville—presented papers of unusual interest and of pertinent value to the physician in general practice, as well as to those in the more specialized fields. Practically every speaker illustrated his talk with lantern slides, which is always an effective means of emphasizing one's topic.

One hundred fifty M.D.'s registered for the two-day Assembly but a number of others were observed in attendance who had not registered. A total registration of 290 covered also the Technical Exhibitors, wives, and members of Academies of General Practice from neighboring States.

The Board of Directors held its Annual Meeting on Wednesday night, preceding the opening of the Assembly, at which a volume of important business was transacted. Having voted at its December, 1951, meeting henceforth to hold the Academy's Annual Business Meeting, with election of officers, directors, etc., during its Annual Scientific Assembly in May, this meeting took place on Thursday, May 8, at which time the officers and chairmen of committees made their annual reports and officers, directors, and delegates and alternates to the House of Delegates of the American Academy of General Practice were elected.

Chosen as President-Elect for the year 1953-54, was Dr. Brewster A. Hopkins of Stuart; Vice-President, Dr. Richard M. Reynolds of Norfolk; re-elected as Secretary and Treasurer, respectively, were Drs. W. Linwood Ball of Richmond and Clifton R. Titus of Bedford; Directors elected to represent the Second, Fifth and Eighth Congressional Districts, in which terms of office of Directors will expire in October, 1952, were: Drs. Harry M. Frieden of Norfolk, John J. Neal of Danville and Charles W. Warren of Upperville. Dr. Mary Elizabeth Johnston of Tazewell was re-elected Delegate to the House of Delegates of the National Academy, with Dr. George P. Hand, Jr. of Norfolk as Alternate. Dr. W. Linwood Ball was elected General Chairman of the Annual Scientific Assembly for 1953 and directed to appoint his own Business Manager, the time and place of which meeting will be the second Thursday and Friday in May, at the Hotel Jefferson in Richmond.

The Woman's Auxiliary met at Luncheon on Friday, which was largely attended, a feature of which was an interesting demonstration on flower arrangement. A number of wives added their names to the steadily growing membership of the Auxiliary and the incumbent officers were re-elected to serve another year.

The Assembly was preceded on Wednesday, May 7, by an afternoon meeting of the Virginia Chapter of the American College of Chest Physicians. A most interesting program had been arranged, which was attended by a goodly number of the General Pracitioners, who reported the papers presented outstanding in their interest and value.

The Board of Directors of the Academy unanimously voted to invite the Virginia Chapter of the American College of Chest Physicians, as well as the Diabetic Association, to schedule their annual program meetings to coincide with the Annual Scientific Assembly of the Virginia Academy of General Practice throughout the future and to integrate their programs with those of the Academy, if they so desire.

Highlight of the two-day Assembly was the social event which concluded the program—the Annual Banquet and Dance—which was preceded by a delightful cocktail party, given through the courtesy of Van Pelt and Brown, Inc. A brief but pertinent and much enjoyed and appreciated address by Dr. John T. T. Hundley, Jr., President of The Medical Society of Virginia, followed the Banquet, during which vocal and instrumental entertainment had been provided.

The guest speaker was Dr. Alfred P. Haake of Park Ridge, Illinois, economist, lecturer and consultant to the General Motors Corporation, through

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whose courtesy he addressed the Academy. Dr. Haake's subject was "An Economic Diagnosis of America", which truly brought out the ills of our national economic set-up and program and pointed out the great need for a return to the integrated home life of our forefathers and to the religion which was a part of their daily life. Illustrating his remarks with many "homey" incidents which struck a responsive chord, he drove forcefully home the need for an awakening to the subversive elements in our home and national life and for a revival of the fundamental principles of home and community life, based upon Christian principles, and an abiding and steadfast faith and belief in the efficacy of God. His address left his hearers spellbound and with a feeling of having listened to profound truths, which, if followed, could produce a nearer approach to the "Utopia" which all human beings seek, whether consciously or unconsciously.

The dance, which followed shortly after the conclusion of Dr. Haake's address and which was interspersed with a good floor show, was a delightful climax to a most beneficial and enjoyable two days.

H.M.S.

Progress Report on Crippled Children's Services Coordinated by the Virginia Council on Health and Medical Care

One of the brightest spots on the Virginia health scene in recent years is the work being done in the field of crippled children. Here The Nemours Foundation requested that the Virginia Council on Health and Medical Care sponsor and coordinate the work with handicapped children so that The Foundation could extend their aid to Virginia. At The Foundation's request the Council held a Conference in September 1951 for the purpose of calling together all the various groups and individuals in the State which work with handicapped children to acquaint them with the services available at the present time. The following types of handicapping were discussed in detail: Speech and Hearing, Orthopedic Conditions, Cerebral Palsy, Epilepsy, and Rheumatic Fever and Cardiac Conditions. Over 100 different organizations, both State-wide and local, official and unofficial, participated. Numerous interested individuals came also. As a direct result of this first Conference The Nemours Foundation is putting $45,000 into our State's program during 1952. The Council has set up a Coordinating Committee on Crippled Children's Services to carry out conference recommendations and formulate a plan which will lead to greater team-work among the groups serving our handicapped children.

The Council is now proceeding to plan a second Conference. It will be devoted to Speech and Hearing which was one of the phases of handicapping considered at the general conference last year. The meeting will be held at the University of Virginia, Thursday and Friday, September 11th and 12th. All physicians in the State are invited and urged to attend. A preliminary program and other information can be secured by addressing inquiries to the Virginia Council on Health and Medical Care, 102 East Franklin Street, Richmond 19, Virginia.

The following are some of the groups which work with speech and hearing and which will be participating: Bureau of Crippled Children, State Health Department; Special Education and Vocational Rehabilitation, State Board of Education; Virginia Cerebral Palsy Association; Virginia Society for Crippled Children and Adults; Virginia School for the Deaf and the Blind; Virginia State School; Junior League School of Speech Correction, Richmond; Speech Clinic, University of Virginia; Hampton Roads School of Speech; Virginia Society of Otolaryngology.

We hope that our second Conference will afford an opportunity for physicians, official and voluntary health workers, and others, to learn about the facilities available and services offered to children with speech and hearing defects. With this information in mind or available in a directory, it is hoped that more children with these two handicaps can and will be referred to the agency, clinic, or center where adequate and proper treatment can be given. We also hope that as a result of the coming Conference plans can be worked out to improve the future care and treatment of children with speech and hearing defects, by having a better coordinated program for the State. The possible advantages of such a program will be many.

It is hoped that the physicians of the State will take advantage of the Conference on Speech and Hearing, and plan to attend.

EDGAR J. FISHER, JR., Director
Virginia Council on Health and Medical Care
Richmond, Virginia

MENTAL HEALTH

JOSEPH E. BARRETT, M.D.,
Commissioner, Department of Mental Hygiene and Hospitals

Nursing Service in State Mental Hospitals*

The concept of care for the mentally ill in primitive civilization was based on the belief that mental disease was caused by evil spirits taking possession of the body of an individual in punishment for the sins committed by that individual.

Treatment of mental illness was directed towards driving out these demons and many brutal measures were employed for this purpose.

In the early Christian Era prayer and the laying on of hands by the priest were used to exorcise these demons and when this failed offensive language, punishment and torture were employed. In England in 1537 an institution used to receive "lunatics" was called "Bethlehem Asylum", which later became known as "Bedlam", and here attendants were allowed to charge an admission fee of a penny for the exhibition of mental patients.

In the seventeenth and eighteenth centuries we find voices raised against inhumane treatment of the insane and such persons as Philippe Pinel, William Tuke and Benjamin Rush successfully demonstrated that kindliness and diversion could take the place of restraint and brutality.

American concepts of treatment for the insane were those that the colonists brought with them and followed the pattern of treating insane persons as prisoners. The teachings of Benjamin Rush, that occupational therapy could be employed in the care of the insane had a great influence on medical thought and practice.

Eastern State Hospital, Williamsburg, built in 1773, was the first hospital in America used exclusively for the care of the insane, and similar hospitals were erected in other states.

In 1843 Dorothea Lynde Dix, who had been visiting jails, workhouses, and places of detention for insane patients, found conditions so deplorable that she presented to Congress the results of her survey which resulted in the state's making appropriations for the building of suitable hospitals and passing laws to protect the insane.

The standard of care conformed to the belief that insane patients were dangerous to themselves and others, would always remain so, and should be locked away safely.

Later developments in the nineteenth century were the organization of training schools for nurses or attendants in mental hospitals and Miss Linda Richards, America's first trained nurse devoted many years to organizing nursing schools and teaching in mental hospitals.

The Mental Hygiene Society, organized in 1908 by Clifford Beers, a recovered mental patient; the books which have been published by patients who have recovered from mental illness; the impact on the public of the realization of the large numbers rejected by Selective Service because of emotional instability; the casualties resulting from mental and nervous disorders during the Second World War; and the overcrowding of our mental hospitals, have awakened the public to the fact that mental illness is a public health problem of major importance.

The recognition of this problem by Congress resulted in passage of the National Mental Health Act in 1946, which provided for development of a long range nation-wide program for improving the mental health of the nation. Because of the shortage of psychiatrists, nurses, social workers, psychologists and others, one of the first objectives of this program is the preparation of an adequate number of workers in the field of psychiatry.

What about the treatment of mentally ill patients in the hospitals which are financed by the state?

Exposé of conditions in some of these hospitals in recent years have shown many deplorable condition resulting from overcrowding, lack of adequate personnel and insufficient funds to provide minimum standards of care.

Recent study and research have shown that mentally ill patients cannot only lead more comfortable lives within the hospital but that the use of modern psychiatric treatment and techniques can result in the early return of many patients to the community as useful and productive citizens and indeed by instituting treatment before the patient reaches the

*This article prepared by Miss Annie A. Hall, R.N., Psychiatric Nursing Education Director, Department of Mental Hygiene and Hospitals, Commonwealth of Virginia.

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stage of hospitalization he can be prevented for entering the hospital.

It would seem, therefore, that the treatment provided for mental patients should not be gauged by the number of available dollars but rather on the basis of what is needed to carry on a program of treatment directed towards recovery. Can we afford to do less when we consider the increased population within mental hospitals during the past twenty years?

What is the adequacy of the nursing service within our state mental hospitals?

The standards for psychiatric hospitals and clinics approved by the American Psychiatric Association, November, 1951, recommend:

One registered nurse to five patients, and
One attendant to four patients in the admission and intensive treatment service of the mental hospitals;
One registered nurse to 40 patients, and
One attendant to six patients in the continued treatment service;
One registered nurse to 20 patients, and
One attendant to four patients in the geriatric service;
One registered nurse to five patients, and
One attendant to five patients on the tuberculosis service;
One registered nurse to five patients, and
One attendant to five patients on the medical and surgical service.

While the ratios of nurses and attendants to patients may seem high at first glance, when considered in the view of a 40-hour and 5-day week they present a modest recommendation.

No public mental hospital has yet achieved these *standards*, Dr. Leo M. Bartemeir, president of the American Psychiatric Association, conceded. "But", he said, "if we really want to give mental patients active treatment and humane care—and who doesn't —then there is no excuse for pussyfooting about what is required to do the job." "After all", he said, "the public has long since demanded and taken for granted high standards in our general hospitals. Can we any longer deny our mentally ill the same consideration?"

Administrators in our mental hospitals have been greatly concerned over the inadequate number of well prepared personnel within the nursing services, to carry out an effective program of treatment and care of patients. Most of these hospitals have only a few graduate nurses, so that the care of the patient has been almost entirely entrusted to the attendant and there are too few attendants. It would seem that since the attendant is so vitally related to the care of the patient, he, more than any other, is the one the patient sees; he is the one who, while others may be able to await the proper time to make a good approach, must see that the patient eats *now*, even when food is refused. He must see that acceptable standards of personal hygiene are maintained, and, in short, must see that the patient is kept alive as well as be there to counsel, guide and befriend him. He should be selected for his desirable qualifications for the work, given a careful training for his duties, and receive the salary and recognized status to afford him continued satisfaction in his work.

Frequently this has not been the case. The meagre salary paid the attendant has made it difficult to attract sufficient people of the right calibre to this work, so that, when economic conditions outside the hospital are good, many vacancies exist and often the problem of ward coverage is so acute that it has been difficult to give proper consideration to qualifications when employing attendants. This contributes to the unsatisfactory attendant-patient relationships which at times are brought to the attention of the public.

According to the Committee on Careers in Nursing, there was a shortage at the beginning of this year of 65,000 graduate nurses in civilian fields and it is unlikely that the shortage will be relieved for some time. During 1951 the nursing schools operated at about 88% of capacity with around 7% of high school graduates entering the field. From these figures it seems doubtful that mental hospitals can in the near future, even if finances allow it, be staffed with well prepared graduate nurses. Graduate nurses are, and always will be, essential for organizing, directing, teaching and supervising the nursing services and educational programs.

In many states various programs are being developed to provide well prepared personnel in the nursing services. The "attendant" who has been closely associated with custodial concepts of care is now being replaced in many states by the "psychiatric aide". He is a member of the nursing service who has been carefully selected and prepared to carry out, with others of the team, the modern care and treatment of the mental patient. In the Department of Mental Hygiene and Hospitals, Commonwealth of Virginia, there exists at the present time many

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vacancies for graduate nurses and for attendants. The need for a better prepared nursing service is a recognized one inasmuch as it is realized that future progress in the care of the mentally ill will be in relation to the adequacy of the nursing service to carry out the program of treatment directed by the physician.

Educational programs within the nursing service of our Virginia State Hospitals are being developed as follows:

(1) An in-service program for graduate nurses who have not had previous psychiatric preparation in nursing. Such a program is now in operation at Central State Hospital, Petersburg, Virginia.

(2) Expansion of an affiliation in psychiatric nursing within all state mental hospitals. At the present time only our Eastern State Hospital has an affiliation in psychiatric nursing.

(3) A course of advanced preparation in administration and supervision in psychiatric nursing.

(4) A program for a new classification of nursing personnel to be known as "Psychiatric Aide".

Recognizing that, for the present, sufficient graduate nurses to staff our hospitals are not available and that the present classification of attendant has failed to attract sufficient numbers of persons of the right calibre to fill existing positions for attendants, we propose to establish the position of psychiatric aide.

The psychiatric aide will be required to have a high school education, or have the ability and background to meet the equivalent of a high school education. He will be selected for his emotional stability, personality, interest and aptitude for this work; will be given adequate preparation for his duties, and should receive the salary and recognition necessary to attract qualified persons to this work as a career.

This course for the psychiatric aide has as its aim: "To provide a group of personnel within the nursing service, who have the necessary understanding, knowledge and skills to contribute to effective care and treatment of the mentally ill in our hospitals, and to the promotion of better health in the community."

Advanced courses for psychiatric aides who are to become charge psychiatric aides and supervisors will be developed, as well as advanced courses for graduate nurses, for, only by continued preparation for duties and responsibilities can graduate nurses and psychiatric aides be effective as members of the team working towards the recovery of the patient.

New Physical Laboratory.

Latest addition to AMA headquarters in Chicago is the new physical laboratory which was opened last month for testing of devices submitted to the Council on Physical Medicine and Rehabilitation. Dr. Frederick T. Jung, director of the laboratory, says that the majority of the laboratory's work is concentrated on testing actual mechanics of new devices submitted by manufacturers to the Council. This supplements the clinical testing which will continue to be done by practicing physicians who cooperate with the Council in this way. The results of physical and clinical testing are referred to the Council for evaluation and approval.

New Director Heads Medical Education Foundation.

The Board of Trustees has announced the appointment of a full time executive secretary for the American Medical Education Foundation. Mr. Hiram W. Jones, former director of finance for the Chicago region of the National Conference of Christians and Jews, assumed his duties with the Foundation May 1. An intensified campaign among state societies for voluntary contributions for the Foundation is being conducted this spring. Russell F. Staudacher, who formerly directed the activities of both the American Medical Education Foundation and the Student American Medical Association, will devote his full time as executive secretary of the Student AMA.

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PUBLIC HEALTH

MACK I. SHANHOLTZ, M.D.,
State Health Commissioner of Virginia

The Cancer Control Program

Cancer stands second as the cause of death in the United States, ranking next to deaths from cardiovascular diseases and arteriosclerosis. There are well over 200,000 deaths per year and there are approximately 700,000 persons afflicted with cancer at any one time. In Virginia there were 3,324 deaths from cancer in 1951.

Because of the magnitude of the problem and the fact that it affects almost all of our families, fifty-two states and territories have established Cancer Control programs. These facilities and services are supported by State and Federal funds. The official cancer programs are administered by some unit of the health department in all of the states and territories except Arkansas and New Hampshire where a state cancer commission has the responsibility.

The Bureau of Cancer Control of the Virginia State Health Department came into being as a result of the interest of the Virginia branch of the American Cancer Foundation, now known as the American Cancer Society, and the Cancer Committee of The Medical Society of Virginia which resulted in the passage of a resolution by the General Assembly of Virginia in February, 1944, directing the Virginia Advisory Legislative Council to make a study and report on the problem of cancer control and treatment in Virginia. The Council made its report to the Governor in September 1945, and, as a result, the General Assembly of 1946 made an appropriation to the State Health Department for use in cancer control in the state during the following biennium. This appropriation together with a grant of Federal funds made through the United States Public Health Service, resulted in the establishment of the Bureau of Cancer Control of the Virginia State Health Department in 1947.

The program as established is the program that is being carried on to-day. Its major element consists of case-finding with emphasis on early diagnosis and prompt and adequate treatment. Case-finding is the result of efforts by individuals, private physicians, dentists, public health nurses, and detection clinics. Diagnostic studies are made by private physicians, dentists, hospitals in connection with examinations for other diseases, and diagnostic clinics. Certain supplementary projects have been undertaken to implement the above and these include epidemiological studies, educational programs, both professional and lay, biopsy service, and aid to clinics.

The epidemiological studies are studies of the background of the development of cancers rather than the causes. This is done by abstracting the hospital records of tumor cases throughout the state and from these establishing a Central Cancer Register in the State Health Department. These records will be subjected to statistical study.

The educational program is extremely vital. Professional education is carried on in cooperation with the Cancer Committee of The Medical Society of Virginia and lay education in cooperation with the Virginia Division of the American Cancer Society. Professional education consists of talks to professional groups, presentation of films, distribution of "The Cancer Bulletin" to the doctors of Virginia registered with the State Board of Medical Examiners, and cooperation with the two medical schools. Lay education is carried on through addresses to women's clubs, civic clubs, parent-teacher associations, films, and workshops.

The biopsy service offers to the physicians of Virginia for their medically indigent Virginia patients free examination of biopsies when sent to the participating pathologists accompanied by the forms supplied by the Bureau of Cancer Control. The biopsies are sent by the physician direct to the pathologist and the reports are made direct to the physician. A copy is sent to the Bureau of Cancer Control. A list of the cooperating pathologists, the necessary forms and mailing cases may be obtained from the State Health Department, Bureau of Cancer Control.

Aid to clinics is in the form of an honorarium to one clinician per clinic session in each of the tumor clinics approved by The Medical Society of Virginia and the Hospitalization-Diagnostic Service. This permits the approved tumor clinics to admit for hospitalization for a period not exceeding three days certain patients on whom they find that further study

for diagnosis will be necessary. The State Health Department does not enter into the payment for treatment of cancer patients.

The whole cancer control program of the State Health Department has been approved by The Medical Society of Virginia. The Cancer Committee, one of the permanent committees of The Medical Society of Virginia, acts as an advisory committee for things pertaining to cancer control in the state. In supporting activities of the Bureau of Cancer Control of the State Health Department members of The Medical Society of Virginia are lending their support to action taken by the Cancer Committee and the House of Delegates of their own Society.

MONTHLY MORBIDITY REPORT OF THE BUREAU OF COMMUNICABLE DISEASE CONTROL

	April 1952	April 1951	Jan.-Apr. 1952	Jan.-Apr. 1951
Brucellosis	3	4	7	19
Diarrhea & Dysentery	82	122	1015	651
Diphtheria	9	8	36	52
Hepatitis	32	1	243	5
Measles	3543	3074	9657	7164
Meningitis (Meningococcic)	27	10	78	52
Poliomyelitis	0	1	8	14
Rabies in Animals	53	22	221	55
Rocky Mt. spotted fever	1	0	3	0
Scarlet fever	117	100	330	578
Tularemia	0	1	22	18
Typhoid & Paratyphoid	4	2	18	18

BOOK ANNOUNCEMENTS

The Fight Against Tuberculosis. An Autobiography. By FRANCIS MARION POTTENGER, M.D. Henry Shuman, Inc., New York, N. Y. 1952. xii-276 pages. Cloth. Price $4.00.

Diagnostic Bacteriology. A Textbook for the Isolation and Identification of Pathogenic Bacteria. By ISABELLE GILBERT SCHAUB, A.B., Technical Director, Clinical Bacteriology Laboratories, The Johns Hopkins Hospital; etc. And M. KATHLEEN FOLEY, M.A., Instructor in Bacteriology, Department of Biological Sciences, College of Notre Dame of Maryland; etc. Fourth Edition. St. Louis, The C. V. Mosby Company, 1952. 356 pages. Cloth. Price $4.50.

Living in Balance. By FRANK S. CAPRIO, M.D. The Arundel Press, Inc., Washington, D. C. 1952. x-246 pages. Cloth. Price $3.75.

Histopathological Technic. Including a Discussion of Botanical Microtechnic. By ARAM A. KRAJIAN, Sc.D., Formerly in Department of Pathology, Los Angeles County General Hospital, Los Angeles, Calif. And R. B. H. GRADWOHL, M.D., Pathologist to Christian Hospital, St. Louis; etc. Second Edition. St. Louis, The C. V. Mosby Company. 1952. 362 pages. With 131 Text Illustrations and 7 Color Plates. Cloth. Price $6.75.

Penicillin Decade. 1941-1951. Sensitizations and Toxicities. By LAWRENCE WELD SMITH, M.D., Medical Director, Commercial Solvents Corporation, and ANN DOLAN WALKER, R.N., Former editor "Trained Nurse and Hospital Review". Arundel Press, Inc., Washington, D. C. 1951. 122 pages. Price $2.50.

An up to date review of the published reports of untoward reactions occurring during penicillin therapy. In conclusion, the author issues a word of caution against the indiscriminate use of antibiotics. With complete bibliograph.

J.D.R.

Biological Antagonism. The Theory of Biological Relativity. By GUSTAV J. MARTIN, Sc.D., Research Director, The National Drug Company, Philadelphia. The Blakiston Company, Philadelphia and New York. 1951. xii-516 pages.

Much of the literature pertaining to the general problem of antagonism in biology has been brought together in this book. Although not entirely complete in certain aspects, the main purpose of compiling a reference work has been well accomplished. Naturally, the pharmacological effects of many substances have been discussed. Besides consideration of drug action, chapters are also included reviewing the action of amino acid, protein, vitamin, purine, pyrimidine, lipoid, and ion antagonists. An attempt has been made to relate the antagonistic action of these substances to a competition with naturally occurring metabolites for certain specific reaction sites. Certain further interesting speculation has been presented, but the author has been careful, in so far as possible, to separate his own ideas from the main body of facts.

S.S.

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WOMAN'S AUXILIARY TO THE MEDICAL SOCIETY OF VIRGINIA

President ..... MRS. HERMAN W. FARBER, Petersburg
President-Elect—
MRS. THOS. N. HUNNICUTT, JR., Newport News
Recording Sec'y..... MRS. L. BENJ. SHEPPARD, Richmond
Corresponding Sec'y.. MRS. CARNEY C. PEARCE, Petersburg
Treasurer ..... MRS. KALFORD W. HOWARD, Portsmouth
Publication Chairman MRS. ROBT. H. DETWILER, Arlington

National Meeting.

The Woman's Auxiliary to the American Medical Association has issued an interesting program for their twenty-ninth annual meeting in Chicago, June 8-13. Headquarters will be at the Conrad Hilton Hotel (formerly the Stevens). Tickets for all general sessions and social functions of the Auxiliary should be obtained at the time of registration. Registration desk is in the Grand Ballroom Foyer (Mezzanine Floor) of the Conrad Hilton Hotel.

The general program will start on Monday, the 9th, at 9:00 a.m., with round table discussions, and the formal opening of the annual meeting will be at the same hour on Tuesday. The general meetings and entertainments will run through Thursday, the 12th. Mrs. Harold F. Wahlquist of Minneapolis, president, will preside.

News from Auxiliaries.

WARWICK

At the meeting of the Auxiliary on February the 27th, Mr. Ford of Langley Field, by invitation, spoke on "Survival Under Atomic Attack". It was announced that there was a First Aid class taught by the Red Cross, with ten members of the Auxiliary taking instruction.

Dr. and Mrs. J. F. Gayle announce the birth of a daughter on March the 9th, and Dr. and Mrs. C. W. Beaven also on March the 10th.

The March meeting was a luncheon at Hotel Warwick in Newport News on the 26th, at which plans were completed for Doctors' Day which was observed on the 30th of that month.

CAMMIE E. DICK (MRS. MURRAY),
Chairman, Publicity.

NORTHAMPTON-ACCOMAC

The Spring meeting of this Auxiliary was held at the home of Mrs. J. Fred Edmonds in Accomac on April the 8th with thirteen members present. The members enjoyed a dessert course and a social period, following which the president, Mrs. John Wise Kellam, presided at the business session. Following the devotional, minutes of the January meeting were read and the treasurer's report given.

Mrs. Kellam reported on the meeting in Richmond and urged members to stress civilian defense programs.

Members were asked to aid in the nurse recruitment program and in the entertainment of student nurses. They were also urged to take a more active part in politics.

Mrs. J. L. DeCormis asked that members give as much publicity as possible to The Gulfstream Nurseries' open house day for the benefit of the Northampton-Accomac Hospital. It was also suggested that members attend "off the shore" meetings when possible.

Mrs. John Hamilton, chairman of "Today's Health", requested that members promote this publication, both among the laity and the medical profession.

The Auxiliary will be guests of Mrs. John Wise Kellam at her cottage at Silver Beach on the second Tuesday in July.

CATHERINE R. TROWER (MRS. E. HOLLAND),
Chairman, Press and Publicity.

EDITORIAL

Nannie Jacquelin Minor, R. N., 1871-1934

IN 1900, when Miss Nannie Minor graduated at the Old Dominion Hospital Training School, nursing, as indeed all medicine, was poorly organized in Virginia. Most of the patients in the hospitals were surgical and most of the surgery was appendectomy. Well do we remember when we returned to school after a short vacation home, being asked, by Stephen Watts, who was resident on surgery, if we saw any surgery other than appendectomies. Richmond had no full time health officer and tuberculosis led by a wide margin all diseases in mortality. Typhoid fever and infantile diarrhea were endemic.

The first training school for nurses in this country was established at Bellevue in 1873 and in the same year New Haven, the Massachusetts General Hospital, and Johns Hopkins also started training schools. The first training school in Virginia was St. Luke's where, in 1886, "Miss Walker, a clever and accomplished lady, presides over the nurses", who included seven ladies and one or two male nurses. "The course of instruction included lectures, demonstrations, and recitations in anatomy, physiology, symptomatology, therapeutics, and hygiene." In 1891 a training school for colored nurses was organized at the Hampton Training School. The Retreat for the Sick started its training school in 1893 and the Virginia Hospital one in 1895-96.

In 1895 the Old Dominion Hospital started a two year course of nurses' training under Miss Sadie Heath Cabaniss, a graduate of the Johns Hopkins Hospital School. She is usually considered the first superintendent of a training school in Virginia, and she certainly is due great credit for the high plane of nurse training in Richmond.

Such was the hospital and nursing background at the turn of the century. A few hospitals had short courses to train scarcely sufficient nurses for their own use. Other hospitals had no such training and there was only one hospital training school comparable to the training schools of the North.

Nannie Jacquelin Minor was born June 15, 1871 to John B. Minor and Anne Colston Minor, the youngest of a family of two brothers, two sisters and a half sister. John B. Minor was the first professor of law at the University of Virginia and probably the greatest in the whole South. Her mother was an invalid and it has been suggested that this was the determining factor in her dominating desire to become a nurse. She was a delicate child and did not go to school, but had her father's niece as a governess. Whether this was on account of the social status or of her health, the record is not clear. The only other medical factor in her upbringing was that both Dr. Osler and Dr. George Ben Johnston were friends of the family.

Considering the newness of the nursing profession and the fact that it had not won its place in the esteem of the public, together with Miss Minor's poor health characterized by nervous vomiting, the opposition of her family to her entering a training school is understandable. Nevertheless she went forward with her plans and wrote to the Johns Hopkins Training School for application blanks. As it turned out she entered the hospital as a patient rather than as a probationer. Evidently she was suffering from anorexia nervosa, for she was given the treatment usually accorded such patients at that time—isolation and observation. At the end of the "cure", Dr. Osler advised the family that the patient should be allowed to enter the training school.

It seems that Miss Minor had been a patient in the Old Dominion Hospital before she was sent to Johns Hopkins and had become fond of Miss Sadie Cabaniss who had

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been brought to Richmond to establish a training school modeled after the one at Hopkins. Although she had carried an application to the Johns Hopkins Training School for three years, she decided on Miss Cabaniss' school. Later she took some post-graduate courses at Johns Hopkins and the Thomas Wilson Sanatorium.

In 1900 Miss Cabaniss, Miss Minor, Miss Sarah Harvie, and Miss Randolph rented a house in the poor section of town and started a Nurses' Settlement patterned after the Hester Street Settlement which had been established in New York some ten years previously. This was the starting point of social work in Richmond. They were at once confronted with three problems: to pay the rent, to "sell" the idea to the citizens of Richmond, and to demonstrate to their neighbors how to live hygienically. In the first group there were eight nurses who worked after hours. They tried to interest the various churches, but had little success.

The social and semi-social agencies of the time were the City Mission, the City Board of Health (no full time health officer), the King's Daughters, Children's Aid Society, Public Charities, whose superintendent was also in charge of the City Home, Little Sisters of the Poor, Hebrew Benevolent Association, Lee Camp Auxiliary, Methodist Mission, Ice Mission, Y.W.C.A. and the Y.M.C.A. The work of this social group soon came to the attention of Mrs. B. B. Valentine who asked Miss Minor to tell the Woman's Club about their work. The result was that the ladies brought some business into the organization. The Instructive Visiting Nurses Association was incorporated and a charter was obtained in 1901. Miss Arents gave them the use of a house, rent free for three years. Miss Cabaniss was titular head of the Association, at first, but by common consent most of the executive problems fell on Miss Minor's shoulders.

For funds to carry on the work, the I.V.N.A. was dependent upon gifts. The Metropolitan Life Insurance Company paid them a fee for each visit to a policy holder. After 1908 an annual tag day was held. Whenever they had more money than they actually needed, they would add an extra nurse to take care of their most pressing need as a pilot project. Some of these would be taken over by the city, and the I.V.N.A. would turn to another project. The first of such projects was a tuberculosis clinic which was started in the home of a widow on Maiden Lane. This was an immediate success and the Health Department took over the work and established two clinics in different parts of the city, and the I.V.N.A. started another pilot project at the suggestion of Dr. William Tate Graham. This grew into the Crippled Children's Hospital. Each summer they ran a babies' hospital at Lakeside for Dr. McGuire Newton until health conditions in Richmond became so good that the hospital was no longer needed. School nursing was another pilot project. This was taken over by the school board. Well baby clinics is one of the projects that the I.V.N.A. has continued in their district while the city Health Department has carried on the work in other parts of the city. The same may be said of the prenatal clinic. The home delivery service is one that the I.V.N.A. has held on to from the very beginning. It fits so well with their neighborhood classes, bedside nursing, loan closet et cetera.

In addition to her work with the I.V.N.A., Miss Minor is to be remembered for her work with the Nurse Practice Act in 1903, and she was a member of the original State Board of nurse examiners, and a charter member of the Graduate Nurses Association of Virginia.

In 1921, Miss Minor resigned as director of the I.V.N.A. to take charge of the organization of State Public Health Nursing Service, a position she held until 1932.

Miss Minor died in Lewisburg, W. Va. on January 30, 1934, and is buried in the cemetery of the University of Virginia. Her grave is marked by a modest stone which bears the simple inscription "Virginia Pioneer of Instructive Visiting Nursing".

Original Articles for Publication

A CONSIDERATION of the present backlog of articles awaiting publication in the VIRGINIA MEDICAL MONTHLY discloses that at least a seven month's supply is now on hand. The interval of time between the submission of an article and its publication varies during the year and is closely related to the flood of articles presented at the annual meeting of the Medical Society.

It would seem desirable to achieve publication of an article within six months after it is submitted. Obviously such an ideal is not always possible but, in order to maintain a reasonably short interval, the editorial board must give consideration to the length of articles. Because of the limitations of space, those exceeding six pages in the MONTHLY must await their turn in a separate category. Ordinarily it is not possible to publish more than one long article each month. Thus, if an unusual number of long articles is on hand, a shorter article may be pushed ahead to an earlier publication date. This policy seems only fair in order to serve the largest number of authors.

A study was made of the length of original articles published during 1950 and 1951. In 1950 there were seventy short articles of four pages or less, fifteen medium length articles of five to six pages, and fourteen long articles of six pages or more. The figures for 1951 were sixty-three, twenty-eight and eight respectively.

Authors are urged to conserve space by every means possible. Only those x-rays should be selected for publication which can be expected to reproduce satisfactorily. A long and detailed list may often be presented more effectively by composite graphs or by summaries. By this method not only is a vast amount of space saved but the reader's attention is more readily attracted. Lengthy bibliographies probably have little place in a State Medical Monthly. L.H.B.

Floral Eponym

HEBENSTRETIA

Hebenstreit, Johann Ernst, 1703-1757, studied medicine in Leipzig but, before completing his thesis, he made a journey of scientific exploration to Africa. Upon graduating in medicine he entered the faculty and for the last ten years of his life he was dean. He died December 5, 1757.

Hebenstretia is a genus of 30 species of South African herbs or shrubs.

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PRESIDENT'S MESSAGE

ELSEWHERE in this issue of the MONTHLY there is an editorial by Dr. Vernon Lippard concerning the American Medical Association Education Foundation. I hope it will be read carefully by every member of the Medical Society of Virginia.

Nothing more vitally concerns the medical profession of America today than the support of the medical schools in this period of their great crisis. The freedom of American medicine is as immediately threatened by governmental control of medical education as by the institution of compulsory health insurance.

If private means are not immediately found to provide financial support to the medical schools of this country, the only recourse will be to accept financial aid from the Federal government, and that very quickly means control by the Federal government.

A committee has been appointed in Virginia to raise funds for medical education in cooperation with the American Medical Associaton Education Foundation. Dr. Marcellus Johnson, Jr., of Roanoke is chairman. You will be hearing from that committee in the near future. Be prepared to give liberally, that you may maintain your freedom.

JOHN T. T. HUNDLEY, M.D., *President,*
The Medical Society of Virginia

SOCIETIES

The Spring Clinical Conference

Of the Midtidewater and Northern Neck Medical Societies was held at Tappahannock in the Yacht Club, Dr. A. Broaddus Gravatt, presiding. The program was conducted under the auspicies of the Committee on Continuation of Medical Education of The Medical Society of Virginia, of which Dr. Kinloch Nelson is chairman.

The program was of practical value to the general practitioners assembled.

Dr. M. J. Hoover, Jr., Medical College of Virginia, spoke on "Office Treatment of Common Fractures."

Dr. E. L. Kendig, Richmond, on "Infant Feeding."

Dr. C. G. Gibson, Medical College of Virginia, spoke on "Antibiotics, the Uses and Selection in Treatment of Various Diseases."

The next meeting of the Midtidewater Medical Society will be held at Urbanna, July 22, 1952.

The Fairfax County Medical Society

Met at the home of Doctor Nelson Podolnick in Falls Church on April 10th.

There was a very short business meeting, followed by a paper by Doctor Sigmund Newman on "Limitations of X-Ray". There was an unusually large attendance. The meeting adjourned for refreshments.

The next meeting will be held on the second Tuesday in May at the home of Doctor William Harris.

ALICE H. KIESSLING,
CLAUDE COOPER,
Publicity Committee

Alexandria Medical Society.

Officers of this Society to serve for the year beginning May are: President, Dr. Milton R. Stein; vice-president, Dr. F. Preston Titus; and secretary-treasurer, Dr. John E. Roberts. Dr. Jerome Baum was elected a member of the Executive Committee for two years. Drs. Walter L. Nalls and A. C. Wyman continue on the committee for another year.

The Lynchburg Academy of Medicine.

The April meeting of the Academy on the 14th was held at a dinner at the Virginia Hotel, Lynchburg, but no business of importance was transacted at this time.

Richmond Academy of Medicine.

At the meeting of the Academy on May 13, the guest speaker was Dr. Alto E. Feller of the Department of Microbiology of the University of Virginia. His subject was "Streptococcal Infections and the Prevention of Rheumatic Fever".

NEWS

Scientific Exhibits.

Dr. Eugene L. Lowenberg, Chairman of the Committee on Scientific Exhibits, announces that the Committee will now receive applications for scientific exhibits for the meeting of The Medical Society of Virginia to be held in Richmond, September 28th through October 1st. The deadline for applications will be June 15th. The executive committee will then choose those to be shown and notify the exhibitors by July 1st. This will be necessary because of limited space at disposal for scientific exhibits. Applications should be requested from Dr. Hunter B. Frischkorn, Jr., 1000 West Franklin Street, Richmond 20, Virginia.

News from the State Health Department.

Dr. V. A. Turner has resigned as assistant director of local health services to accept a position with the Mountain Home Veterans Hospital in Johnson City, Tennessee.

Dr. James M. Suter,

Who has been health officer in the Smith-Washington-Bristol district with headquarters at Abingdon, has been appointed by State Health Commissioner, Dr. M. I. Shanholtz, as assistant director of health services in Southwest Virginia, succeeding Dr. V. A. Turner, resigned. He will continue to operate from Abingdon.

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The Virginia Academy of General Practice

Held its second annual Scientific Assembly in Roanoke, May 8 and 9. This was a most interesting and successful meeting and is reported in detail in the Miscellaneous Department of this issue.

Dr. Thomas K. McKee Honored.

The Smyth County Medical Society held its meeting on April 16, in honor of Dr. Thomas K. McKee of Saltville, who is retiring from part of his duties after fifty-five years of practice in Smyth County. A banquet was held for him at the Lincoln Hotel in Marion. Dr. J. C. Motley of Abingdon was the guest speaker and presented Dr. McKee with a key from the County organization in recognition of his long and faithful service to the medical profession. The banquet was largely attended by a number of doctors and their wives.

Dr. Charles E. McKeown,

Richmond, announces the removal of his office to Lee Medical Building, Monument Avenue at Allen.

TV to Bring AMA Meeting to Public.

Doctors who cannot personally attend the American Medical Association's annual meeting June 9 to 13 in Chicago will have the opportunity of viewing its highlights via television. As the nation's top medical authorities report to doctors on the latest medical developments, NBC mobile TV units will move through the corridors of the exhibit hall at Navy Pier, transmitting the news coast-to-coast. Present arrangements call for two half-hour programs to be televised on Tuesday and Wednesday evenings over most NBC network stations. The telecasts are being sponsored by Smith, Kline and French, Philadelphia pharmaceutical firm.

Dr. and Mrs. Philip Jacobson,

Petersburg, left early in May for Europe, where Dr. Jacobson will attend sessions of the International College of Surgeons at Madrid. They will return about the first of July.

Dr. Harvey B. Haag,

Professor of pharmacology at the Medical College of Virginia, Richmond, was selected as president-elect of the American Society for Pharmacology and Experimental Therapeutics at its forty-second meeting held in New York City in April. Doctor Haag was secretary of this organization from 1947 to 1951. Only once before has its presidency been held by a scientist from a southern institution.

The primary purpose of the society is to further research on the physiological effects of chemicals of importance to human health, particularly in terms of their discovery and use as drugs in the prevention and treatment of disease. It was founded in 1908 by Dr. John J. Abel, late professor of pharmacology at the Johns Hopkins University School of Medicine.

Gill Memorial Spring Congress.

The Twenty-fifth Annual Spring Congress of the Gill Memorial Eye, Ear and Throat Hospital was held in Roanoke, Virginia, April 7 through 12, 1952. This was the Silver Anniversary of the Congress and it marked the largest attendance in the history of the school. The total registration including ladies was 464. The 1953 meeting will be held April 6 through 11.

News from University of Virginia Department of Medicine.

Dr. William Parker Anslow, Associate Professor of Physiology at New York University, has been appointed Professor of Physiology and Chairman of the School of Physiology. He will succeed Dr. Sydney W. Britton who will retire in June following a leave of absence as Fulbright Fellow in South Africa. Dr. Anslow has been associated at New York University with Dr. Homer Smith, a former Professor of Physiology at the University of Virginia.

Dr. William Edward Bray will retire as Professor of Clinical Pathology and Director of the Clinical Laboratories in July. With the exception of one year, he has served the Department of Medicine continuously since receiving his M.D. degree from the University in 1912, and has taught thirty-nine classes.

Dr. Bray first organized the clinical laboratories at the University in 1922. He founded the school for medical technologists at the University Hospital and compiled useful laboratory techniques and procedures into "Clinical Laboratory Methods", a manual now in its fourth edition.

The scientific program of Medical Alumni Day, June 6, will be opened by Dr. J. William Hinton, Professor of Surgery of New York University Post-

Graduate Medical School, who will speak on "Physiologic Factors in the Management of Essential Hypertension". Members of the faculty of the University Department of Medicine will give reports of studies in progress at the medical school.

Dr. Harry B. Taylor, President of the Medical Class of 1902 which will celebrate its fiftieth anniversary this June, will address alumni and graduates of the Class of 1952 at the Annual Alumni Dinner, Farmington Country Club, June 6.

Eli Lilly and Company Aids Flood Victims.

Eli Lilly and Company is replacing all Lilly products in pharmacies and hospitals ravaged by the flood in the Missouri and Mississippi River Valleys. Representatives in a dozen states, from Montana to Missouri, have been directed to make the replacement of flood-damaged Lilly pharmaceuticals and biologicals their first order of business. Along with the replacement of stocks, the Lilly company maintains a reserve supply of typhoid vaccine and other biological products which is kept ready for fast shipment during disasters. The Company has been replacing stocks damaged by uninsurable hazards as far back as the 1906 San Francisco disaster.

Drs. Rawles and Cherry.

Dr. Benjamin W. Rawles, Jr., and Dr. Kenneth J. Cherry announce the removal of their offices to 2306 Monument Avenue, Richmond.

The American Society of Medical Technologists

Will hold their twentieth annual convention in Portland, Oregon, June 22-26. All members of the medical profession will be welcome.

Society for the Prevention of Asphyxial Death.

To the first 100 Virginia physicians who become members of the Society following this notice, the offer has been made to donate a copy of the Art of Resuscitation by Paluel J. Flagg, M.D., autographed by the author. The book is listed for $6. Physicians who wish to receive this book, however, are asked to apply for membership in the Society, enclosing membership dues of $5. Communications should be addressed to Secretary, S.P.A.D. Inc., 2 East 63rd Street, New York City 21.

Dr. Gill Honored

At the annual meeting of the New York Eye and Ear Infirmary in New York City on April 22nd, 23rd, and 24th, Dr. E. G. Gill of Roanoke, was elected president of the Alumni Association of the New York Eye and Ear Infirmary and also a member of the Board of Directors of the Hospital.

Editorial Assistants-Collaborators.

A clearinghouse service on competent editorial assistants or collaborators to assist in the preparation of papers for meetings, publications or clinical demonstrations is being established. Technicians qualified to assist in editing explanatory or sound tract material in conjunction with professional motion pictures are included. For information address Academy-International of Medicine, 214 West Sixth Street, Topeka, Kansas.

Guided Tours of AMA Headquarters.

If you are planning to visit Chicago for the AMA meeting, plan to take advantage of the new guided tour service at AMA headquarters. This service will be available during the AMA annual session, and all attending the convention are invited to visit 535 North Dearborn Street. Guided tours will leave the AMA front lobby every hour on the hour from 9 a.m. to 4 p.m., June 9 to 13 . This tour program is to be a permanent AMA service.

1952 Award Contest.

The National Gastroenterological Association again announces its Annual Cash Prize Award Contest for 1952. One hundred dollars and a Certificate of Merit will be given for the best unpublished contribution on Gastroenterology or allied subjects. Contestants residing in the United States must be members of the American Medical Association.

All entries for the 1952 prize should be limited to 5,000 words, be typewritten in English, prepared in manuscript form, submitted in five copies accompanied by an entry letter, and must be received not later than 1 September 1952. They should be addressed to the National Gastroenterological Association, 1819 Broadway, New York 23, N. Y.

Wanted—

Associate physician (general practice); drawing account plus commission; rapid advancement; twenty miles from Richmond. Write P. O. Box 257, Providence Forge, Va. Phone 541. (*Adv.*)

XUM

Medical Resident Wanted.

Beginning July 1, fully approved 165-bed general hospital has opening for Medical Resident. Stipend $150 a month and maintenance. Address "Medical Director", C. &. O. Hospital, Huntington, West Virginia. (*Adv.*)

Wanted—

Experienced Superintendent of Nurses and Laboratory Technician for a small hospital. A satisfactory salary with maintenance. Lebanon General Hospital, Lebanon, Virginia. (*Adv.*)

Wanted—

General Practitioner in Virginia, to practice with small established group. Address "Physician", care Virginia Medical Monthly, 1105 West Franklin Street, Richmond 20, Va. (*Adv.*)

General Practitioner

Licensed in Virginia desires to purchase home and practice in Tidewater area. To replace, or join with established physician, or will consider entering into an area of need. Reply "No. 90," care this journal, 1105 West Franklin Street, Richmond, Va. (*Adv.*)

OBITUARIES

Dr. Matt Otey Burke,

Retired physician of Richmond, who had practiced medicine for more than sixty years, died at his home here on April 23. He received his degree in medicine from Tulane University in the class of 1891 and practiced in Russell County before locating in Richmond. He was connected with the staffs of the former University College of Medicine and later the Medical College of Virginia for some years. His wife and three sons, one of them Dr. James O. Burke, survive him.

Dr. Russell Landram Haden,

Crozet, died April 26 in Cleveland, where he, accompanied by his wife, had gone to attend a meeting of the American College of Physicians. He was sixty-four years of age and a graduate in medicine from Johns Hopkins University in 1915. He was a noted authority on blood diseases and rheumatic diseases and at the time of his death was national chairman of the Red Cross blood program. He had been a member of The Medical Society of Virginia since returning to Virginia.

Dr. Charles M. Hazen,

Who practiced in Richmond for many years until his retirement, died at a nursing home in Bon Air, April 22, after a long illness. He was eighty-seven years of age and graduated in 1897 from the Medical College of Virginia, in which he was emeritus professor of Physiology.

Dr. Bolling Jones Atkinson

Died at his home at Emporia, April 15. He was fifty-two years of age and a graduate of the Medical College of Virginia in 1923. He had been a general practitioner in Greensville County for twenty-seven years and was formerly a member of The Medical Society of Virginia. His wife and two children survive him.

Dr. William Miller Dick,

Of Hampton, was found dead in a hotel room on April 22. He was fifty-one years of age and a graduate of the Medical College of Virginia in the class of '26. He had been a member of The Medical Society of Virginia for several years. His wife and two children survive him.

Dr. William B. Hopkins,

Tampa, Florida, died suddenly April 27, of a heart attack. He was a Virginian by birth and graduated from the former University College of Medicine in Richmond in 1907. He practiced in Richmond for sometime before moving to Florida where he had made his home for the past twenty-five years. His wife and two children survive him.

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